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PRACTICAL LECTURES

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PRACTICAL LECTURES

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THE MEDICAL SOCIETY OF THE COUNTY OF KINGS
BROOKLYN, NEW YORK

[1923-1924 SERIES]

ONE HUNDRED AND THIRTY-TWO ILLUSTRATIONS
AND THREE COLOR PLATES

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LIST OF CONTRIBUTORS

BLAKE, JOSEPH A., M.D.
Surgical Director, Reconstruction Hospital; Consulting Surgeon, St. Luke's, Roosevelt, Orthopaedic, Beekman St. and Lincoln Hospitals, New York; St. John's Riverside Hospital, Yonkers; Dobbs Ferry and Tarrytown Hospitals, N. Y.

BRAY, HARRY A., M.D.
Medical Superintendent, New York State Hospital for Incipient Pulmonary Tuberculosis, Ray Brook, N. Y.; Adjunct Assistant Visiting Physician, Second Medical Division (Cornell) Bellevue Hospital, New York City.

BROOKS, HARLOW, M.D.
Professor of Clinical Medicine, New York University; Visiting Physician, City Hospital, New York City.

CAMPBELL, WILLIAM FRANCIS, A.B., M.D., F.A.C.S.
Clinical Director of the Brooklyn Cancer Research Hospital.

DEAVER, JOHN B., M.D.
Emeritus Professor of Surgery, University of Pennsylvania; Surgeon-in-Chief, Lankenau Hospital, Philadelphia.

DICKINSON, ROBERT L., M.D.
Senior Gynecologist and Obstetrician, Brooklyn Hospital; Consultant Obstetrician, Methodist Episcopal Hospital, Brooklyn, N. Y.

EWING, JAMES, M.D.
Professor of Pathology, Cornell University Medical College, New York City.

FORDYCE, JOHN A., M.D.
Professor of Dermatology and Syphilology, College of Physicians and Surgeons, Columbia University; Visiting Dermatologist, City Hospital, New York City.

Fowler, Russell S., M.D.
Chief Surgeon, Wyckoff Heights Hospital; Surgeon, Methodist Episcopal Hospital, Brooklyn, N. Y.

Gant, Samuel G., M.D.
Attending Coloproctologist to the Broad Street and Harbor Hospitals; Consultant to the Joint Disease, Huntington, Jewish and Memorial Hospitals, New York City, to Hackensack Hospital and to Sing Sing Prison.

Jennings, John Edward, M.D., F.A.C.S.
Surgeon, Brooklyn Hospital.

Johnson, Louis C., M.D.
Assistant Professor of Medicine, Long Island College Hospital, Brooklyn, N. Y.

Libman, Emanuel, M.D.
Physician to Mount Sinai Hospital; Professor of Clinical Medicine, Columbia University, New York City.

MacAusland, W. Russell, M.D.
Surgeon-in-Chief Orthopedic Department, Carney Hospital, Boston; Medical Director Berkshire School for Crippled Children, Pittsfield, Mass.

McCrae, Thomas, M.D., F.R.C.P. (London)
Professor of Medicine, The Jefferson Medical College, Philadelphia.

Mosenthal, Herman O., M.D.
Professor of Medicine and Attending Physician, New York Post-Graduate Medical School and Hospital.

Oliver, Wade W., A.B., M.D.
Professor of Bacteriology, Long Island College Hospital, Brooklyn, N. Y.

Polak, John Osborn, M.D., M.SC., F.A.C.S.
Professor of Obstetrics and Gynecology, Long Island College Hospital; Director of the Department of Obstetrics and Gynecology, Long Island College Hospital, Brooklyn, N. Y.

Regan, Joseph C., M.D.
Physician in Charge of Contagious Diseases, Department of Health, New York City.

TILNEY, FREDERICK, M.D.
Professor of Neurology, Columbia University, New York City.

TRUSLOW, WALTER, M.D., F.A.C.S.
Consulting Orthopedic Surgeon, Brooklyn Hospital, Victory Memorial Hospital, Seaside Hospital, Far Rockaway, N. Y.

WALSH, JAMES J., M.D., PH.D., SC.D.
Medical Director, Fordham University School of Sociology; Professor of Physiological Psychology, Cathedral College, College of the Sacred Heart, Manhattanville, N. Y.

WARREN, LUTHER F., M.D.
Professor of Internal Medicine and Chief of Medical Service, Long Island College Hospital, Brooklyn, N. Y.

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FOREWORD

THE events leading up to the publication of this volume are unique. In 1922 the Medical Society of the County of Kings celebrated its one hundredth anniversary. To the president, Dr. Frank D. Jennings, there seemed no better way to mark the centennial than by a demonstration of public service. Believing that continuous education of the practicing physician would be the greatest possible contribution that the Society could make in the cause of public health, a committee was appointed to execute the idea.

It seemed that our conventional monthly program, always so carefully planned and up to the minute in progressive medicine, was often inadequate in that it failed to deal with the common problems of everyday practice.

The Committee therefore aimed to give the profession of Brooklyn that which the average doctor thought he needed most. It was decided to hew straight to the line of fundamentally sound teaching, carefully avoiding the realm of theory and experiment. With this in mind the Practical Lecture Series was arranged.

The topics of the lectures were carefully chosen, usually by the chairman of the committee. The speaker was impressed with the fact that he was addressing an audience of general practitioners, not a body of specialists, and that he was to give a talk in every sense of the word rather than read a formal paper. Many of the lectures were illustrated with lantern slides, moving pictures or blackboard demonstrations.

It is believed that this type of "practical" lecture was an innovation. From the start its success was phenomenal. The McNaughton Auditorium in the library building was often overcrowded and on several occasions hundreds of physicians were turned away for lack of room. Since Brooklyn's experiment, other centers have taken up the idea.

Great care was exercised in the selection of speakers. Most of the lecturers were teachers and, where this was not the case, the speaker was selected on account of exceptional work done along special lines. Many talks were offered but were not accepted because the speakers insisted on selecting topics which the committee felt would have too special an appeal and could not fairly be classed as "practical."

After the lectures had been continued with increased popularity, there was a demand for their publication in book form. It was felt that they would prove of great value to men who had not been able to attend. Publication was made possible by the financial assistance of the Joint Committee on Graduate Education of the Medical Society of the County of Kings and the Long Island College Hospital.

The task of seeing these lectures through the press was one not anticipated by the editors when the work was begun. The courtesy, cooperation and support of our authors has been an invaluable help. Credit must also be given to the publishers and their editorial staff, whose experienced assistance has rendered the task a pleasure.

On account of the broad scope and variety of subjects covered in this volume and the "practical" method of handling each topic, it is thought that this book will be found radically different from the average medical publication, and it is felt that it will be a valuable addition to the practitioner's working library.

CHARLES A. GORDON
THURSTON S. WELTON
Editors.

BROOKLYN, N. Y.
November, 1924.

MAR

PRACTICAL LECTURES

THE SURGICAL ABDOMEN*

JOSEPH A. BLAKE, M.D.

THE subject of the surgical abdomen is an enormous one to cover, but it seems that we should make a diagnosis within an hour if we had a patient before us, so I shall hope at least to tell you, in the time allotted me, how we may recognize a surgical abdomen.

By a surgical abdomen I imagine that we mean an abdomen that demands surgical interference. How shall we recognize it? We have certain symptoms and signs. The symptoms are subjective; i.e., what the patient feels. The signs are what we elicit in our examination.

One of the cardinal symptoms, perhaps the most important, and usually the first, is pain. Now, how is pain in the abdomen caused? I have been accustomed, in teaching my students (and in teaching them I have learned much more, I think, than I could have otherwise) to make them try to reason out the various causes so that they would not have to learn them, but would simply come to them in natural processes of thought.

In the first place, pain may be caused by irritation of the sympathetic system, and when so produced is essentially reflex in character. This type of pain is usually violent and is brought about in two chief ways: One, by acute distention of hollow organs, such as the appendix, the gall-bladder or the kidney. The pain is actually caused by violent, spasmodic contractions of the intestines, which in turn are induced by the irritation of the sympathetic system. Understand, the contractions are not peristaltic, but irregular, spasmodic contractions. The second way in which these reflex contractions are caused is by interference with the circulation of organs, more particularly of the intestines; and a typical example of this is the pain caused by strangulation ileus. The pain produced in this way is always severe, and there is

*This lecture was delivered on March 16, 1923.

the same violent contraction of the intestines. In other words, in strangulation ileus, the pain in the early stages is not caused by the obstruction to the flow of intestinal contents, but to the reflex violent contraction of the intestine, caused by interference with its circulation. The proof of this lies in the fact that spasmodic contractions are produced in coils remote from the site of obstruction and in the stomach.

In a second group, pain is caused by irritation of the peritoneum, such as is produced typically by perforation of a gastric ulcer when the intensely acid gastric contents are poured out into the abdomen. The pain thus produced is very severe. Irritation of the peritoneum and pain may also be caused by the extravasation of other fluids, such as urine, feces and blood, but the pain is not so severe, since the irritation is not violent, as that produced by gastric contents. Then we have the pain caused by irritation of the peritoneum, produced by a local peritonitis. This is not the violent, spasmodic pain caused by irritation of the sympathetic system, but a sort of soreness or local pain. It verges, really, on another symptom, namely, tenderness, and is elicited perhaps by the movements of the patient, as in respiration, etc. The pain of peritonitis is usually local. When we have an advanced peritonitis, that is, a generalized peritonitis in the advanced stages, there is no pain; so that it is not simply inflammation of the peritoneum which produces the pain.

Then we have a third important group of causes of pain, namely, local lesions, or the lesions of local organs—as, for instance, gastric or duodenal ulcer. Usually, the local lesion produces a pain which has its own characteristics. There is a pain with cholecystitis or gall-stones, usually attributed to indigestion. This is rather undefinable, and is very difficult to place under any single head, as it may be reflex produced by distention of the gall-bladder, or caused by local irritation of the peritoneum. Of course, the tenderness in cholecystitis is due to the latter cause.

Then we have a fourth group of causes of pain, namely, the conditions that produce hyperperistalsis, the overaction of the intestines. The pain produced by hyperperistalsis is

usually known as gas pain. A typical example is produced by an obturation ileus, that is, an intestinal obstruction by stoppage, or by plugging from within. A more common cause is irritating intestinal contents, such as produce cholera morbus. The pain of hyperperistalsis differs in degree from those produced by the violent spasmodic contraction of the intestines accompanying sudden distention of an organ, or strangulation ileus.

Then we have a number of other causes which produce pain. These are not altogether surgical, but they have to be eliminated in a diagnosis. Such are the pain of tabetic crises, of angiospasm, and the pain caused by angioneurotic edema, any of which may simulate severe surgical lesions. I have also observed abdominal pain in patients suffering from general purpura. There is also the pain of lead colic. We do not know exactly to what this pain is due, but it is probably caused by toxic irritation of the sympathetic system, producing violent spasmodic contractions of the intestines, similar to those mentioned before as of sympathetic origin.

The second cardinal symptom which we have to consider is vomiting. We find that many of the causes which I have mentioned as produce pain also cause vomiting. In the first place, we have irritation of the sympathetic system produced, as stated before, by the sudden distention of organs, such as the gall-bladder, the appendix or the kidney. We have seen that pain, produced by irritation of the sympathetic system, was in all probability directly caused by the violent spasmodic contractions secondary to such irritation. Vomiting, as observed in these cases, may also be considered as due to contractions of the stomach, set up in the same way. We all know that with the sudden onset of violent colicky pain, we usually have vomiting, and the two symptoms may be taken as a fairly accurate index as to the amount of distention that has occurred in some viscus. I consider it a point well worth remembering, that when vomiting ushers in an attack of appendicitis, it means that there has been sudden violent distention of the appendix, and, therefore, an emergency exists.

As a second cause of irritation of the sympathetic system, we have the interference with the circulation of the intestine produced by strangulation. We have all observed, in cases of

strangulation, say, of the ileum, that the patient has a violent pain—sympathetic irritation—and vomits several times, the vomitus consisting of stomach contents only. Then there is a period, if the case is allowed to proceed, in which there is no vomiting and in which the violent primary pain gradually subsides; symptoms of peritonitis supervene, as gangrene ensues, and finally symptoms of obstruction appear and obstructive vomiting occurs; but it is late. For example, suppose we take strangulation of the lower part of the ileum. In this condition, vomiting from back filling, that is, filling up of the intestine backwards, does not occur for several days, and yet the intestine may have been gangrenous since early in the attack.

So there are two kinds of vomiting in intestinal obstruction. Here, again, as in the case of the distended appendix, sudden and early vomiting means a critical situation. Violent pain plus vomiting always indicates a serious situation. Of course, we may have both pain and vomiting with sudden distention of the kidney, but the situation may not be so serious as in the case of the appendix or gall-bladder, for we haven't the danger of peritonitis supervening.

A second major cause of vomiting is intestinal obstruction of the obturation type. In this variety of obstruction, the vomiting, or the time of onset of the vomiting, depends largely upon the site of the obstruction, but it is relatively late. Of course, an obstruction at the pylorus produces rather early vomiting, the vomitus consisting of stomach contents. In obturation of the colon, vomiting may not occur for a week or more. We have to wait until the intestine fills up, then the stomach, and the vomiting is simply the emptying of the overfilled organs. This sequence of events is observed whether the obturation of the intestine is produced suddenly, as by a large gall-stone, or slowly, as by a carcinoma. It is interesting that vomiting does not occur unless the obturation by stricture, neoplastic or cicatricial, is complete. As I have already mentioned, in strangulation ileus, vomiting is caused secondarily by obstruction at the site of strangulation.

There is another kind of obstruction that produces vomiting, namely, the obstruction we call adynamic or paralytic ileus, which is due to paralysis of the muscularis of the intes-

tine and is observed in peritonitis, chiefly in its advanced stages. The dilatation of the stomach, which is seen most commonly in peritonitis or after operations, with or without peritonitis, belongs in this category. Of course, after every operation there is some peritonitis, but dilatation of the stomach may occur with negligible peritonitis.

We may have vomiting from the very common condition of overloading the stomach, or from an indigestion, or with cholera morbus, just as we may have pain in cholera morbus from hyperperistalsis.

In the fourth group, we have as causes of vomiting, conditions of toxic origin, such as from alcohol, drugs and poisons, uremia, pregnancy, etc.

Another subjective symptom is shock. This has no particular diagnostic value as to determining what or where the lesion is. It is rather an evidence of a disaster or a very critical condition, as when the patient goes into shock after the perforation of a typhoid ulcer.

We now come to the second group elicited by ourselves, namely, the objective signs. I think the first and most important of these is rigidity of the abdominal wall. You may call it rigidity or spasm. I have always used the term rigidity. I remember one of the first papers I wrote on the abdomen was on the diagnostic significance of abdominal rigidity. It may seem strange, but in medical school I was never taught anything about abdominal rigidity and did not know anything about its importance. I had to find it out for myself, so I thought it was worth while making it the subject of a paper.

The most marked examples of abdominal spasm are those caused by severe irritation of the peritoneum, such as extravasation of gastic juice or of urine or feces and even of blood. The abdomen in a severe case is, as you know, scaphoid—its walls are drawn in. It seems as if the retraction was a protective spasm to prevent the motion of the intestines beneath the abdominal wall. We also see it in the early stages of peritonitis, when it is far more marked than in the late stages. I think you have all observed that, in late peritonitis, particularly postoperative peritonitis, we do not find true spasm. The muscles are tired out, apparently, and everything then

seems to be relaxed. There is a certain amount of resistance, which I shall speak of later, but not the true spasm that denotes something violent going on within the abdomen. Rigidity is quite marked in cases of pancreatitis because there is a very active irritation of the peritoneum, as well as peritonitis, in some cases of acute pancreatitis.

I have also observed quite marked rigidity of the abdominal wall, so much so as to lead me into the error of operating, in cases of pneumonia with diaphragmatic pleurisy. One should always be on his guard for these cases. They are usually diagnosed by other signs and symptoms, but even if we examine for rigidity alone it will be found that the rigidity is more marked in the upper abdomen, the abdomen below being a little softer. The drawing in of the muscles is also more noticeable, perhaps on one side, with a non-excursion of the diaphragm and lack of movement of the ribs on that side, indicating that a thoracic condition is combined with the abdominal condition. Now, these patients have a true abdominal condition. They have a peritonitis, as well as pleurisy. Two such cases were operated upon by me and in both I found a peritonitis involving the upper abdomen—a distinct peritonitis, reddening, exudate, etc., but it was simply secondary to the infection just above the diaphragm, or rather an extension of this infection.

We also have another group of causes which produce rigidity, namely, those acting by reflex. Many of the same conditions that produce pain and vomiting through irritation of the sympathetic system, provoke rigidity. For instance, strangulation of the bowel, renal colic and lead colic produce marked rigidity of the abdominal wall. One may easily mistake renal colic for some intraperitoneal condition, because in some cases the rigidity and retraction of the abdominal wall are very marked; and if one operates, as I did once, on such a case, one finds the same spasmodic condition of the intestine that I have spoken of under the causes of pain. The small intestine in the case to which I refer was contracted to the size of a lead pencil and contracted irregularly in different places. In this particular case, not finding any particular cause, I closed the abdomen. There was no peritonitis. The next day the patient passed a calculus.

I have always differentiated abdominal resistance from abdominal rigidity because, to my mind, it conveys other conditions in the abdomen. An abdominal resistance, as distinguished from rigidity, is rather doughy in character, as, for instance, the resistance which we find in the late stages of suppurative peritonitis, or in some cases of chronic peritonitis (more particularly tuberculous peritonitis) and in typhoid fever. In these conditions, there is a resistance, but none of the board-like spasm of the muscles so characteristic of something violent going on within the abdominal cavity.

Tenderness is one of the very important signs in abdominal examination. Tenderness, and particularly the point of major tenderness, gives us a clue to the location of disease. This is particularly so in local peritonitis. For example, it is usually easy to differentiate between cholecystitis and appendicitis by the site of tenderness. It generally gives the clue to disease of the kidney. Yet the appendix may be near the gall-bladder or kidney and so give rise to error. The appendix may lie in the pelvis and the tenderness be low down, simulating that of salpingitis, and yet real tenderness at McBurney's point is an almost infallible diagnostic sign of appendicitis. In cases of extravasation into the peritoneum, tenderness is usually most marked, not at the point of perforation or rupture, but at the points to which the extravasated fluid gravitates and causes the most active inflammation, as, for instance, in the right iliac fossa in perforation of a duodenal ulcer.

The next sign we look for is a mass in the abdomen. This may be caused by a local peritonitis; for example, the thickening about an organ, produced by agglutination of the omentum or otherwise, such as about the appendix or tubes. It may be a distended organ itself, such as a gall-bladder or a kidney. It may be a neoplasm. It may be produced by a fecal accumulation.

Another sign is abdominal distention. Here it is quite important to note the time of its onset and also its location. One of the causes that produces a most marked distention of the abdomen is obstruction of the obturation variety in the lower bowel. The lower the obstruction the greater the

abdominal distention, because the more coils of intestine are above it to be distended. In such a case, the colon is first distended and it may be several days before the small intestine is involved. Volvulus also produces a very marked distention, particularly volvulus of the sigmoid flexure. Distention in volvulus of the sigmoid develops rapidly and involves the lower part of the abdomen first.

Before I leave these causes of distention, I wish to say a few more words about them. In the first place, one cannot be too careful in following the development, or obtaining the history of the development of distention. Suppose, for example, we have a carcinoma of the sigmoid flexure and it becomes obstructed; where will the distention first be seen? I remember once coming over to this city when I was considerably younger, to examine a case for Dr. Walter James. The patient was an old lady, who had abdominal pain situated chiefly on the right side. She had distention of the right side. She had no particular tenderness there; at least, no more than would be elicited by a simple sensitive bowel from overdistention, but the distention and pain were quite marked. In watching the abdomen for a little while, it became evident that there was active peristalsis. The peristalsis was chiefly situated on the left side, while the distention was on the right side; so I said she had a carcinoma of the sigmoid flexure and that that was the region to attack at operation. We operated and found it there. Dr. James said to me, "How could you make a diagnosis of that when the pain was on the other side?" "Well," I said, "In obstruction of the lower part of the colon the distention occurs first in the cecum because its wall is thinner and it distends more readily; and also, the obstruction being of a chronic nature, the muscularis of the intestinal wall immediately oral to the growth becomes hypertrophied and it is almost the last to give way." So it is a good point to remember, to elicit where the distention commences.

In cases of obstruction by volvulus, of course, the sigmoid flexure takes a turn upon itself, and then it commences to fill up and distend with gas. In most of these cases there is more or less of a megacolon; that is, the sigmoid is larger than it normally should be. The distention arrived at in

some of these cases is simply enormous. When the abdomen is opened, the distended sigmoid bulges out, and, the restraint of the abdominal wall being relieved, it will often be some six or eight inches in diameter, and its loop will rise a foot or two above the incision. When you observe the muscularis cracking you must introduce a knife through its wall to relieve the pressure and prevent it from really ripping apart.

Another very marked cause of abdominal distention is paralytic ileus, known also as adynamic ileus of late peritonitis, and particularly postoperative peritonitis. In these cases the distention gets to be very great, as you often see; and the greater part of it is due, as a rule, to distention of the stomach; that is, gastric dilatation. The stomach can usually be felt, or mapped out, easily by percussion, and even its shape may be seen.

Then there may be quite remarkable distention of a toxic nature, such as in pneumonia. We have all seen this.

You may find a moderate amount of distention caused by gas free in the peritoneal cavity. This condition can be diagnosed by the loss of liver dullness. Lastly, there are the cases of distention from fluid exudate in the abdomen, such as in ascites or chronic serous peritonitis.

Another very important sign in reaching a diagnosis is the observation of visible peristalsis. This practically always means that there is an obstruction which the intestine or stomach is endeavoring to overcome. The most marked instances of visible peristalsis are seen in cases of obturation ileus of the large intestine, or even obturation of the small intestine; but the larger peristaltic waves in the large bowel are usually more evident than they are in the small intestine. The maximal peristalsis occurs just proximal to the obstruction. The size of the waves varies according to the size of the organ. The waves in pyloric obstruction due to contraction of the stomach are very easily recognized and occur slowly, passing across the upper abdomen. When peristalsis is visible, it is of the greatest value to listen to the abdomen and find out, if we cannot feel, exactly where the loudest gurgling occurs; so auscultation is a very important aid in examination of the abdomen, particularly

where an obstruction or a peritonitis is to be considered. For instance, one takes a stethoscope and listens, and in this way one can find active peristalsis where none can be felt, or when it is not evident, even to the patient. When the patient speaks of gas rumbling, we know that peristalsis is going on. Peristalsis is an extremely important thing to elicit in making the differential diagnosis between postoperative peritonitis and postoperative obstruction of dynamic character, by which I mean an obstruction which is acting mechanically—either a kink or a twist or some pressure from a drain or otherwise. Quite often the surgeon has to arrive and arrive quickly at a diagnosis between adynamic paralysis of the intestine due to peritonitis and the question of a mechanical obstruction. If there is simply an adynamic obstruction he may decide against operation and use palliative methods, such as washing out the stomach and bowel; but if there is a real dynamic obstruction, he must operate and relieve it. Not long ago, in one of the large hospitals in New York City, I was called in by her friends to see a woman patient, at about the fifth or sixth day after an operation for appendicitis. She had been a healthy, strong woman. It had been an ordinary case of acute appendicitis with a certain amount of diffuse infection, such as we frequently see. It had been closed without drainage. The patient had gone on fairly well and then commenced to distend and have some pain—not much, for pain is not a marked symptom after operation in cases of peritonitis, even when there is obstruction. She commenced to vomit and gradually began to have more and more gaseous distention with more and more vomiting, and the family got worried. They asked me to go in and meet the surgeon who had the case in charge. He said he would meet me, but stated that there was no use in my looking at the patient, as she had a postoperative peritonitis and there was nothing to do. However, I went in and found a man who has quite a reputation for intestinal lavage working with a lot of glass apparatus and tubes. The patient was in a very restless condition, which is always the case with a patient suffering with dilatation of the stomach. In fact, restlessness is one of the first symptoms of the condition. On listening to her abdomen, I could hear peristaltic

waves moving the gas back and forth. My opinion was that the surgeon should operate on her, because I believed she had an obstruction. He did operate and found one, and she got well. The error in diagnosis had arisen from neglect of listening for the signs of peristalsis. If peristalsis is going on, one may be pretty sure that the patient is not dying of an ordinary postoperative peritonitis, at least, not of that alone.

Now, there is another interesting point in the auscultation of hyperperistalsis; and that is in regard to the localization of the obstruction. When the intestine contracts behind an obstruction, the peristaltic waves advance, until the gas and material in the intestine are pushed up against the obstruction. This produces no noises directly, but after they push and push as hard as they can they must give way; and when that occurs there is a backward rush of gas through the advancing peristaltic contractions, so that the transmission of the noises is in a direction backward from the growth. I mean it is oral, that is, toward the mouth, from the growth. This is a very important point to remember in reaching a diagnosis as to the localization of the obstruction.

I have spoken of the chief signs and symptoms elicited by the surgeon. There is another sign which, of course, is very important and which is generally given us by the nurse, and that is the temperature. The course of the fever is all-important in reaching a diagnosis. In certain conditions fever is either moderate or absent. In others it is high; but, as a rule, intraabdominal conditions do not produce very high temperatures. For instance, cases of infection of the gall-bladder with cholectystitis are, as a rule, not attended by a very high temperature. One usually sees it around 100° or 101°F. unless the gall-bladder becomes gangrenous and the condition extends to a peritonitis. Of course, you may have exceptional cases where the fever is high, but, as a rule, it is low. As we all know, it is fairly low in appendicitis. Some exceptional cases of appendicitis, however, are ushered in by a chill and rather high temperature. It does not go much above 101°F. on an average. It often goes higher, of course. We are unable to say that a case is not severe because there is not a very high temperature. We may be, and sometimes are, misled by a low temperature in cases of appendicitis or

gall-bladder disease or renal colic. On the other hand, unless the kidney is infected, there is no temperature. If the kidney is infected and there is a sudden damming back, or a sudden absorption, we may have a chill and very high temperature. The temperature usually goes up higher with infection of a duct system than it does in the intestinal system. For instance, in cholangitis, when the stone goes down, it stops and begins to obstruct, and there is a sudden chill with a very high rise in temperature, which may return to normal if the obstructing stone becomes dislodged and floats back when the duct becomes distended.

As a rule, the degree of temperature helps us to make a differential diagnosis between intraabdominal and intrathoracic conditions, because it is much higher in the pneumonias and in the infections of the chest. In the same way leucocytosis helps us. Polymorpholeucocytosis means infection, of course, and the ratio of the polymorphonuclears to the leucocytes is a pretty good guide to the severity of the infection. Yet, in the very early stages of an acute infection, such as appendicitis, the "poly" count often lingers behind that of the leucocytes.

I have given you the chief symptoms and signs to be elicited in reaching the decision as to whether an abdomen is surgical; and it only remains for me to emphasize the necessity of getting these fixed in mind so that you will be able to use them subconsciously in arriving at a diagnosis. That is to say, the thought runs very much faster than the expression of the thought, and if one groups these various signs and symptoms and looks for them in making a diagnosis, one may often reach what is called a snap diagnosis by very logical processes. That is, if a man has his mind trained to observe these various signs and symptoms, he has only to get the history, and in the examination the diagnosis becomes revealed to him almost subconsciously, because he unconsciously looks for each one of the signs, and if they are not there they are automatically put aside. For example, if we go back for a moment and consider some of the first symptoms I spoke of, namely, the symptoms of pain and vomiting, we will find that when irritation of the sympathetic system is caused and there is marked pain together with marked

initial vomiting, it means either that an organ is very markedly distended, or that there is a strangulation obstruction of the gut. Now, how shall we make a rapid differential diagnosis between them? Subconsciously we jump to the last signs we have considered and ask: "What is the temperature; what is the blood count?" If the patient has a temperature of 99½° or 100° or possibly 101°F., and he has a polymorpholeucocytosis, we rule out obstruction by strangulation. We say it is probably a case of distention of some organ caused by infection, such as appendicitis or cholecystitis. On the other hand, if the blood count and temperature are low, then we are led to look for the signs of strangulation ileus. We look for marked rigidity, etc., and then we think of the various causes of strangulation. The first one of these is a former operation causing adhesions or bands. Or, if the patient be a child, we think of intussusception and so on, and we then commence to listen for peristalsis. In the early stages of strangulation ileus there may not be hyperperistalsis, but, with the development of the signs of obstruction, it comes on and we can hear it. At the time the surgeon approaches the case the diagnostic signs usually have become so pronounced that they attract his attention; and here the surgeon has the advantage of the physician. He not only has the advantage of the physician's history and impressions of the case, but also the development of the case, which the physician had not had at the commencement.

More than once when I have been called in to see a case the first thing that has struck me has been the difficulty of making a diagnosis; and I have often said to myself, "I can well understand how it is that they cannot make a diagnosis," and my sympathy has always been with the fellow that could not arrive at an early diagnosis. It is extremely difficult before the signs are well developed.

There are many patients in whom a diagnosis seems almost impossible. Only last week I saw a patient who had practically the following history: For many years back he had not been well, having had a chronic colon bacillus infection of the genitourinary tract and a chronic colitis. There was a strong suspicion of diverticulitis. This diagnosis had been arrived at largely on account of attacks of abdominal pain and the

appearance of some not very obvious diverticula shown by *x*-ray examination. Well, to be short, his antecedent history, which had been going on for about a year, was suggestive of diverticulitis. One night he began to have sudden violent pain in the abdomen, and with that he vomited and his temperature shot up to about 102°F. He felt as if the bowels must move, which is often the feeling with early spasmodic contractions of the intestines in such cases; the patient often feels that if he could vomit or if the bowels would move he would obtain relief. In the case in question an enema was given, with relief, the temperature came down and the patient was around for three or four days, not feeling perfectly well, but feeling pretty well. Then there was another attack like the first and the surgeon who then saw him said it was the most scaphoid and most rigid abdomen he had ever seen. It was as if it were pulled back to the vertebrae, like a belt, with two bunches above and two bunches below, corresponding to the quadrants of the abdomen. As I have said, this attack was similar to the first, coming on with a chill and a sudden rise of temperature, and then a rapid subsidence of all symptoms. The third attack occurred the night before I saw him, but the pain had not let up. The attack had been ushered in with the same chill and sudden rise of temperature and rigidity of the abdomen. On going over the abdomen, there was not, by rectal or general examination, a spot which one could localize as a point of local tenderness. Yet there seemed to be some intraperitoneal condition. A disturbing thing in making the diagnosis was the localization of the radiation of the pain. The pain was felt most acutely in the rectum and the bladder, and radiated down into the testicle. The picture, then, was a sudden attack, marked by chill, rise of temperature, violent pain in the abdomen extending down to the bladder and rectum and radiating to the testicles, by abdominal rigidity and no localizing point of tenderness. Examination of the urine showed a few pus cells, but no blood. The attack was characteristic of the distention of some hollow organ, probably the kidney, yet relief was obtained in some way by evacuation of the bowel. It was decided to wait until the following morning. The next day there was a distinct mass low down under the right rectus. At operation an

abscess was found between the bladder wall and some coils of small intestine, containing free gas, evidently caused by perforation of the intestine. The sigmoid flexure was in juxtaposition to the abscess and its appendices epiploicae were large and red, but we could not discover any opening. But the next day an intestinal irrigation came out in the wound, showing that there was a perforation of the intestine low down, probably in the upper part of the rectum or lower sigmoid. Now, why was there such a symptom complex with only a localized peritonitis? I may say that the peritonitis must have existed from the first day he had his attacks, because the intestines were so matted together that the coils could hardly be separated. Why, with a localized peritonitis, were the symptoms not continuous? Can the course of the case be explained by an intermittent intestinal obstruction caused by increase in the size of the abscess; or by an intermittent distention of a diverticulum which finally ruptured? The symptoms referable to the urinary tract were evidently due to juxtaposition of the abscess to the bladder.

I recently saw another patient who had been sick nearly a week with violent pain. He had a history (in fact, I had seen him before) of recurrent attacks of abdominal pain for several months. He had been operated upon fifteen years before for suppurative appendicitis and had been drained. In this attack he had violent pain, vomiting and hyperperistalsis, with practically no temperature. On going over his abdomen, not much could be elicited aside from active peristalsis, made out by auscultation. He still had pain, vomiting was letting up and some gas was passing through the bowel and he was evidently getting better. I felt he had had some form of strangulation ileus, but was not sure at first whether he should be operated upon or not. However, on going over his history, we decided he should be operated upon, on account of the repeated recurrence of the attacks and because they were becoming more serious. When the abdomen was opened, we found a cord-like band, the size of a lead pencil, under which the intestine had been caught, as evidenced by marks on the intestine where it had been strangulated, and by signs of local peritonitis.

Generally speaking, where there is violent abdominal pain and vomiting, it is safer to operate. I made this rule long ago. Of course, there are exceptions, but one should take the position that operation is demanded, and assure himself very thoroughly to the contrary if he is going to postpone operation.

AN APPRAISAL OF CERTAIN PHYSICAL SIGNS IN THE DIAGNOSIS OF INCIPIENT PULMONARY TUBERCULOSIS*

H. A. Bray, M.D.

THE physical examination of the chest as a practical procedure dates back to the observations of Laënnec, more than a century ago. Valuable contributions on the subject have since appeared, notably those following Brehmer's demonstration of the curability of tuberculosis. An assiduous search was then begun for evidence which would reveal the disease in its initial or incipient stage, and there are now about one hundred signs said to be of aid in early diagnosis. It is obviously impractical to attempt to master the technique of, and correctly to interpret, so large a number of signs; and only those of practical aid, relatively few in number, have been retained. In this discussion I shall attempt to appraise a few of the signs generally conceded to be helpful in early diagnosis.

Inspection

There are many signs listed under inspection. Let us select for discussion one of the most important: supraclavicular depressions. The value of this sign has been repeatedly emphasized in diagnosis, particularly if the depressions be of unequal depth or unilateral in distribution. In the evaluation of this sign, previous observers have apparently disregarded the fact that similar depressions occur in healthy persons. The recognition of this point is important because if the depressions occur frequently in health, and cannot be differentiated from those in disease, it is obvious that the sign is of little or no value in diagnosis.

Recently, Dr. A. H. Duerschner and myself made a comparative study of the supraclavicular depressions in 153 healthy persons and in 150 patients showing unmistakable evidence of early tuberculosis.

* This lecture was delivered on March 23, 1923.

The healthy series consisted of adults mostly between the ages of eighteen and thirty-five; the proportion of men and women was approximately the same, and a detailed examination failed to reveal any evidence of pulmonary disease. Persons presenting chest asymmetry due to extrapulmonary conditions, spinal curvature, injury, occupation, etc., were not included because they would prove confusing in a study of this nature. We were impressed by the fact that the depressions, occur more frequently in lean than in fleshy persons; and in the same person we observed that the depressions appeared with loss of weight and disappeared when the weight was regained. The depressions are also usually more marked when the muscles about the region of the lung apex are well developed; and this may in part explain the greater depth of the depressions in men than in women, and on the right side in right-handed persons and on the left side in the left-handed.

The size, shape and position of the clavicle influence the depressions, and here the clavicle deserves special mention. For example, in Figure 1, the clavicle is of the long, heavy, straight type, and the outer head is on the same line with the inner head. Under these conditions, the bone is usually prominent, the supraclavicular space is large and the depressions are more or less marked. If, however, the clavicle is of the short type with accentuated curves and the outer head is above and well posterior to the inner head, the bone is generally inconspicuous, the supraclavicular space relatively small and the depressions slight or absent (Fig. 2).

Between these two extreme types of clavicle are found many gradations which explain in part the varied depths of the fossae in health. Even in the same person, the clavicle may show sufficient difference in size and shape to influence the depressions (Fig. 3). Furthermore, regardless of the type of clavicle, it was found that the elevation, or the forward and backward movement of the outer head, changes the depth of existing depressions and may cause their appearance and disappearance. (Figs. 4 *A*, *B*, and *C*). These changes, hinging upon clavicular movement, are important because they explain why a depression present at one examination may be absent at another. Therefore, in confirming observa-

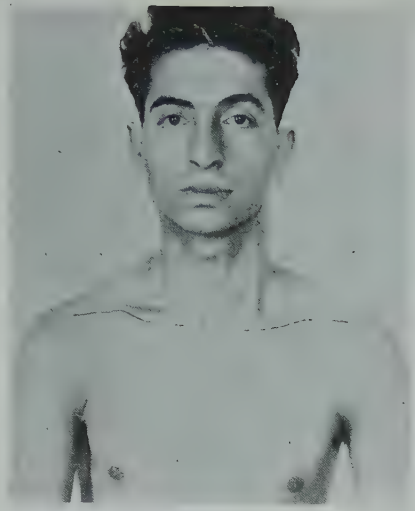

FIG. 1.

FIG. 2.

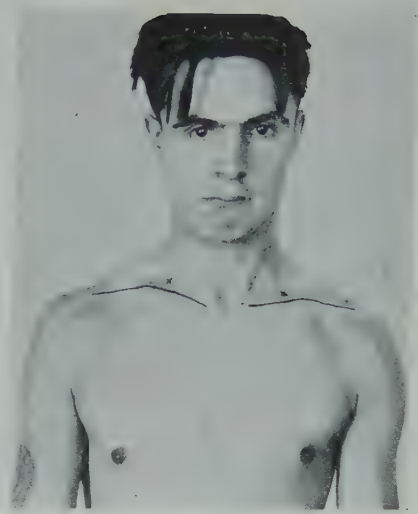

FIG. 3.

FIG. 1. Straight horizontal type of clavicle in healthy person. The outer head of the bone is forward and about on the same line with the inner head. The supraclavicular spaces are relatively large. With this type and position of the clavicle, depressions more or less marked are frequently encountered in health.

FIG. 2. Accentuated curves of the clavicle in a healthy person. The outer head is well posterior to and above the inner head and the supraclavicular spaces are relatively small. In health, depressions are often absent with this type and position of the clavicle.

FIG. 3. Healthy person. Note the difference in the shape of the clavicles, which, in part at least, explains the inequality in the depth of the depressions.

A *B* *C*

FIG. 4 *A*. Healthy subject. Depressions bilateral, equal and of moderate depth.

FIG. 4 *B*. The shoulders are thrown backward and the depressions effaced.

FIG. 4 *C*. The left shoulder has been brought slightly forward, a position which has increased the relative depth of the depression on this side.

tions on supraclavicular depressions, the position of the clavicle should always be identical. It has been suggested that this requisite may be obtained if the patient assume a posture of relaxation. However, one must remember that for every individual there are several postures of equal relaxation, each influencing the position of the clavicle and the relative depth of the depressions.

The above-mentioned factors which influence the depressions are not readily controlled, and the statistical data on the incidence and depth of supraclavicular depressions in health are necessarily only approximately accurate. However, as these factors play a similar rôle in disease, the data may be helpfully employed in appraising the sign in the diagnosis of early tuberculosis.

TABLE 1

THE INCIDENCE AND DEPTH OF SUPRACLAVICULAR DEPRESSIONS IN ONE HUNDRED AND FIFTY-THREE HEALTHY PERSONS

Supraclavicular depressions*		Number	Per cent
Right	Left		
−	−	39	25.4
+	−	27	17.6
−	+	6	3.9
+	+	27	17.6
++	+	15	10.0
+	++	9	5.9
++	++	15	10.0
+++	+	6	3.9
+	+++	3	1.8
++	+++	6	3.9
		153	

* Symbols: −, depression absent; +, depression slight; ++, depression moderate; +++, depression marked.

Table 1 includes the incidence and depth of supraclavicular depressions in 153 healthy persons .tI will be noted that they were absent or negligible in approximately 25 per cent,

while in 75 per cent, depressions were present. In 21 per cent they were unilateral, occurring more often on the right than on the left side. In approximately 54 per cent the depressions were bilateral; of equal depth in 28 per cent, of greater depth on the right in 14 per cent and on the left in 12 per cent.

Table 1 indicates that depressions occur frequently in health; that they are slight or marked, and in distribution unilateral or bilateral; and that their incidence and relative depth is greater on the right than on the left side.

TABLE II

THE INCIDENCE AND DEPTH OF SUPRACLAVICULAR DEPRESSIONS IN ONE HUNDRED AND FIFTY PATIENTS SUFFERING FROM INCIPIENT PULMONARY TUBERCULOSIS

Supraclavicular depressions		Location of lesion					Total	Per cent
		Unilateral disease		Bilateral disease				
Right	Left	Right apex	Left apex	Greater on right	Greater on left	Equally distributed		
−	−	9	6	6	6	3	30	20.0
+	−	9	8	5	5	4	31	20.6
++	−	0	1	2	1	0	4	2.6
+++	−	1	0	0	0	0	1	0.6
−	+	5	1	1	1	1	9	6.0
−	++	1	0	0	0	0	1	0.6
+	+	4	6	3	3	3	19	12.6
++	+	10	8	5	2	3	28	18.6
+	++	5	6	3	1	1	16	10.6
++	++	6	1	0	0	1	8	5.3
+++	+	0	0	0	0	1	1	0.6
+++	++	0	0	1	0	0	1	0.6
++	+++	1	0	0	0	0	1	0.6
		51	37	26	19	17	150	

In the series of 150 cases of early pulmonary tuberculosis in which the depressions were studied, the pulmonary lesion was confined to the apex in all. Patients with definite chest deformity due to extrapulmonary conditions, for reasons previously mentioned, were excluded from this series. The sputum was positive for tubercle bacilli in approximately 32 per cent of the cases. The difference in nutrition between these patients

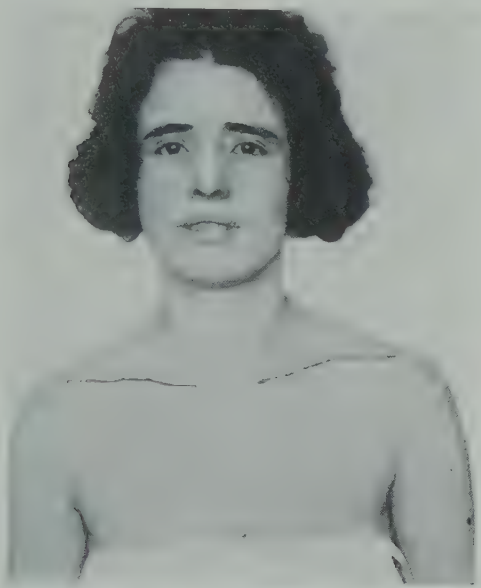

FIG. 5. The pulmonary disease is moderately advanced, bilaterally distributed, confined to the upper lobes and greater on the right side. The depressions are bilateral and of equal depth. From our studies we would be inclined not to attach diagnostic significance to the depressions here present. They occur with the pulmonary lesion but are probably not related to it.

and the healthy group was apparently not sufficient to cause any appreciable difference in the sign.

In Table 11 are tabulated the location of the pulmonary lesion and the incidence and distribution of the supraclavicular depressions in 150 cases of incipient pulmonary tuberculosis. In 20 per cent the depressions were absent; in 49 per cent they were bilateral and in approximately 30 per cent they were unilateral. No evident relation could be established between the depressions and the site of the lesion. This result

was to be anticipated from the striking similarity in the depth and distribution of the depressions in health and early disease, as revealed by a comparative study of Tables I and II.

The pulmonary lesion is not an important factor in producing depressions in early disease, because, even with moderately advanced disease, depressions are frequently absent, and when present, cannot be satisfactorily correlated with the site of the pulmonary lesion, as illustrated in Table III.

TABLE III

THE INCIDENCE AND DEPTH OF SUPRACLAVICULAR DEPRESSIONS IN ONE HUNDRED AND SIXTY-NINE PATIENTS SUFFERING FROM MODERATELY ADVANCED PULMONARY TUBERCULOSIS

Supraclavicular depressions		Location of lesion					Total	Per cent
		Unilateral disease		Bilateral disease				
Right	Left	Right apex	Left apex	Greater on right	Greater on left	Equally distributed		
−	−	2	2	19	15	2	40	23.6
+	−	2	0	14	9	5	30	17.7
++	−	0	0	3	0	0	3	1.7
−	+	0	0	4	6	0	10	5.9
+	+	3	0	13	4	4	24	14.2
++	+	2	1	18	7	3	31	17.8
+	++	0	1	6	2	1	10	5.9
++	++	1	1	7	3	0	12	7.1
+++	+	0	0	0	1	0	1	0.5
+++	++	0	0	0	2	0	2	1.1
++	+++	0	0	1	0	2	3	1.7
+++	+++	0	1	1	1	0	3	1.7
		10	6	86	50	17	169	

Thus, in the patient of Figure 5, although the depressions are of equal depth, the pulmonary disease, in the moderately advanced stage, is definitely greater on the right side; and

in the patient of Figure 6, although the right lung is apparently healthy and the left extensively diseased, the greater depression is on the right (healthy) side.

The earlier observers found supraclavicular depressions a constant sign in pulmonary tuberculosis. At that time, the diagnosis was usually delayed until the pulmonary lesion was advanced and the patient emaciated. They apparently attrib-

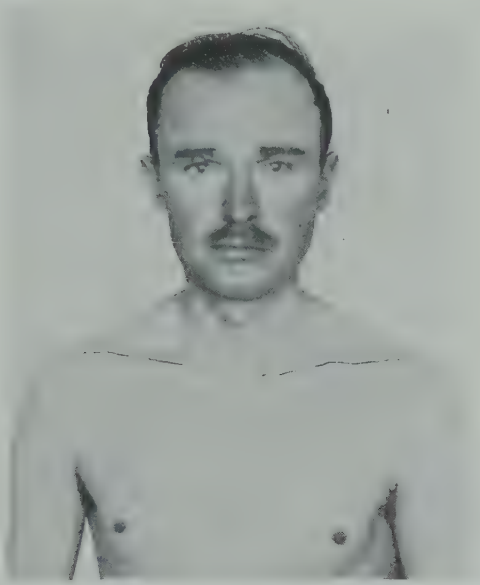

FIG. 6. Bilateral depressions, slightly greater on right. Massive involvement of left lung with antrum formation at the apex. Right lung apparently healthy. In tuberculosis it is not uncommon to find the greater depression on the right side when the lesion is confined to the left lung.

uted the depressions entirely to the underlying lung disease, whereas the major rôle in their production was probably played by the tilting forward of the shoulders from weakness and the associated emaciation. This view is supported by the fact that depressions invariably appear with emaciation, irrespective of its cause. Anatomists, and even dressmakers, have long recognized this relationship. On the other hand, in well-nourished patients suffering from advanced pulmonary tuberculosis, the depressions may be absent.

When depressions have their origin in disease of the underlying lung, they are due to loss of lung volume, associated with local retraction of the chest wall. Local retraction of the chest wall sufficient to influence the depressions occurs for the most part with extensive pulmonary disease and marked loss of lung volume. Even here the presence or absence of depressions depends on the extent to which the individual compensatory factors participate.[1]

In early disease, the loss of lung volume is slight, and is usually compensated for by emphysema or hypertrophy of the lung contiguous to the focus, and, consequently, there is neither local retraction of the chest wall nor change in the soft structures above the clavicle. Therefore, on purely pathologic grounds, one would not expect to find any material change in depressions due to early disease.

Although depression above the clavicle has been regarded as one of the traditional signs for the detection of early tuberculosis, the present study suggests that its value in this respect must be accepted with reserve.

Breath-sound Changes

From a study of tactile fremitus, percussion and breath-sound changes, made at this sanatorium some years ago, we came to the conclusion that these procedures possessed about equal value in early diagnosis. Therefore, the appraisal of one of these procedures, breath-sound changes, will serve as an index of the diagnostic significance to be attached to the other two.

A wide diversity of opinion exists concerning the incidence of the various respiratory alterations and their relative values in diagnosis. Some authorities believe that the granular type of inspiratory murmur is the initial manifestation of the incipient lesion; others emphasize the prolonged high-pitched expiration as indicating early involvement. We studied the frequency and character of the respiratory changes in 124 cases of incipient disease confined to one or both apexes, and the comparative significance of such changes

[1] Bray, H. A. The mechanism of compensation in pulmonary tuberculosis, and its relation to physical signs. *Am. Rev. Tuberc.*, Jan., 1923, vi.

when considered alone and also in conjunction with other physical signs and diagnostic procedures.

Previous investigations included a description of the breath sounds over the entire chest, but our observations were restricted to the apex of the lung. This plan was adopted because it was discovered that a detailed study of the breath sounds over the entire chest was not only an arduous and time-consuming task, but it subjected the patient to an undesirable physical strain. Furthermore, the apexes of the lung are particularly adapted to a study of this kind, since in tuberculosis the initial physical changes occur in these areas in approximately 90 per cent of cases. Finally, by confining the study to patients presenting only apical involvement, and excluding those other conditions which might influence the respiratory murmur in this region, such as disease in other parts of the lung, spinal curvature, deformity of the thorax, obstruction of the upper air passages, etc., it was possible to form an accurate idea of the respiratory variations resulting from apical disease.

The plan adopted consisted in first auscultating the entire chest superficially for the purpose of determining the normal vesicular murmur peculiar to the individual, a requisite for the correct interpretation of pathologic variation in breath sounds. Following this an attempt was made to locate the site of the lesion at the apexes from a study of the respiratory murmur during quiet, then during slightly exaggerated, and finally forcible, breathing, the patient breathing orally and noiselessly and maintaining as far as possible the normal ratio between inspiration and expiration. These findings were then compared with the results of a complete physical examination including the use of the roentgen ray and the fluoroscope. By studying the breath sounds before the other physical signs were elicited, and without knowledge of the roentgen ray and fluoroscopic findings, the examiner was uninfluenced by facts that might tend to color his interpretation of alterations in the respiratory murmur. Previous work had suggested the advisability of this plan.

In this series of 124 cases tubercle bacilli were demonstrated in the sputum of approximately 24 per cent. A comparison of the breath-sound changes in the sputum-

positive and the sputum-negative cases failed to reveal any distinct differences, and for this reason they have been grouped together in the accompanying tables.

The results of the study of the breath sounds in the 124 cases with apical involvement are presented in the following tables. In those cases exhibiting more than one alteration in the vesicular murmur over an apex, only the most marked alteration has been noted.

TABLE IV

FORTY-SEVEN CASES OF INCIPIENT TUBERCULOSIS PRESENTING A LESION AT THE RIGHT APEX

Granular breathing	1
Weakened breathing	5
Increased vesicular	3
Cog-wheel	..
Prolonged expiration	20
Bronchovesicular	6
No change in respiratory murmur	12
Total	47
Breath-sound changes at the normal left apex	8
Weakened breathing	2
Increased vesicular	4
Prolonged expiration	1
Bronchovesicular	1

In Table IV are represented the breath-sound changes in 47 cases of early tuberculosis presenting a lesion at the right apex. In approximately 25 per cent no breath-sound changes were noted, while in the 75 per cent exhibiting respiratory variations, these variations affected for the most part the expiratory and not the inspiratory phase of respiration. It is important also to note that where the other physical procedures failed to reveal evidence of disease at the left apex, the respiratory variations indicated the presence of a lesion at this site.

In Table V are illustrated the respiratory variations in 34 cases of early tuberculosis presenting a lesion at the left apex. The findings were negative in 30 per cent and positive in 70 per cent. In the positive cases, the respiratory variations affected for the most part the inspiratory phase of respiration, which contrasts strikingly with the predominance of

TABLE V

THIRTY-FOUR CASES OF INCIPIENT TUBERCULOSIS PRESENTING A LESION AT THE LEFT APEX

Granular breathing	1
Weakened breathing	10
Increased vesicular	6
Cog-wheel	1
Prolonged expiration	4
Bronchovesicular	2
No change in respiratory murmur	10
Total	34
Breath-sound changes at the normal right apex	9
Weakened breathing	2
Increased vesicular	1
Prolonged expiration	4
Bronchovesicular	2

inspiratory changes in disease at the right apex. In 9 of the 34 cases, variation in the breath sounds indicated disease at the apparently healthy right apex.

TABLE VI

FORTY-THREE CASES OF INCIPIENT TUBERCULOSIS PRESENTING A BILATERAL APICAL LESION

	Right apex	Left apex
Granular breathing	1	0
Weakened breathing	3	5
Increased vesicular	2	
Cog-wheel	1	
Prolonged expiration	12	
Bronchovesicular	4	
No change in respiratory murmur	20	23
Total	43	43

Table VI comprises 43 cases of bilateral disease. In approximately 50 per cent of the cases in this group no respiratory variations were detected, a fact explained largely by the absence of a normal standard for purposes of comparison. In the remaining 50 per cent showing respiratory alterations, it will be noted that the expiratory murmur was more frequently affected at the right and the inspiratory murmur at the left apex. The fact that expiratory changes occur more frequently at the right and inspiratory at the left apex in

early disease, may explain the diversity of opinion regarding the relative diagnostic value of inspiratory and expiratory changes.

The results of the present study do not agree in certain essentials with those of previous observers, notably in the large proportion of cases (approximately 40 per cent) in which the findings resulted negatively. The disparity is explained apparently by the fact that we adopted different methods in their appraisal. This point may be illustrated by selecting for discussion granular breathing, the most important inspiratory variation, and changes in the duration and intensity of the respiratory murmur.

The value of granular breathing has been emphasized by numerous observers, including Grancher, Minor, Turban, Sahli, Otis, Pottenger and Fishberg. For the satisfactory demonstration of granular breathing at the apex, the muscles in this region must be in a state of relaxation, because their active contraction may at times produce sounds indistinguishable from granular breathing, and thus lead to confusion in diagnosis. This requisite is obtained by means of abdominal breathing. In this type of breathing, respiration is conducted solely by the piston-like excursions of the diaphragm, the thorax is fixed, and the muscles in the region of the apex are relaxed.

Abdominal breathing, therefore, represents an efficient method for the elimination of those extrapulmonary sounds which arise in the contracting muscles and which simulate granular breathing. If, however, the granular type of respiratory murmur persists with abdominal breathing, one is justified in concluding that the vesicular murmur has undergone a definite pathologic alteration. The value of abdominal respiration in differentiating extrapulmonary sounds from granular breathing was demonstrated by the fact that in a series of cases previous to its employment, the incidence of this sign in early tuberculosis was 18 per cent, while subsequent to its use, only 3 cases in the present series, or 2.81 per cent, exhibited this sign.

The duration and intensity of the respiratory murmur show considerable variation in health. It is important to recognize the factors responsible for such variations in health because of their frequent confusion with similar pathologic variations. The intensity of the physiologic mur-

mur remains unaltered if the volume of air inspired in a given unit of time is maintained. Thus, if on successive respirations the air intake is 500, 1000 and 1500 c.c. and the time interval occupied by the respiratory act is one, two and three seconds, respectively, no change occurs in the intensity of the respiratory murmur, since the air intake in a given unit of time (500 c.c. in one second) is the same in each instance. The depth of the respiration, however, is altered, as is the duration of the respiratory murmur. The previously accepted view that intensity varies with the depth of respiration is, therefore, open to question. Variations in the intensity of the respiratory murmur are due to differences in the volume of air intake in a given unit of time. The greater the volume, the more intense the murmur, and vice versa. Consequently, the duration of the respiratory murmur depends on the time interval occupied by the respiratory act, its intensity, on the force of the respiratory act.

Physiologic variations in the duration and intensity of the vesicular murmur due to changes in the time interval and force of the respiratory act may be detected by observing the movements of the chest. By the adoption of this procedure the incidence of variations in the duration and intensity of the respiratory murmur were found to be approximately 20 per cent less when compared with those obtained during a previous study.

It will not be necessary at this time to consider the other respiratory variations, because they are of even less value in diagnosis than those here considered. In other words, I do not believe that the so-called respiratory variations noted in early tuberculosis are helpful in diagnosis.

One may naturally ask: "If these traditional signs are of little or no value, what has the physical examination to offer in the detection of early tuberculosis?" I would answer that there is one very helpful sign, and that is the *Rale.*

Rales occur in at least 90 per cent of early cases. In the army this sign was employed as the crucial test in the physical examination to determine whether a man should or should not be drafted into the service. *The diagnosis of pulmonary tuberculosis is justified by the detection of persistent localized rales elicited only by cough, if limited to the upper half of the chest.* In early tuberculosis the rales are peculiar

in that they can be heard on quiet and forced breathing in only 25 per cent of the cases, while in 75 per cent they remain latent until the patient is requested to cough. The position of the cough in the respiratory cycle is highly important for the successful elicitation of the latent rale. For instance, if the patient precedes the cough by a deep inspiration, the cough will be loud, the chest will vibrate, and if the rales are fine and few in number, they may not be heard. However, if the cough is inserted toward the end of expiration, it is reduced in intensity and hacking in character, excellent qualities for the successful demonstration of rales. *The cough should invariably be forceful; a feeble cough may fail to elicit rales.*

It is generally conceded that the origin of the latent rale is in the atelectatic areas surrounding the tubercle, but thus far no satisfactory theory has been advanced to explain its production by the aid of cough. Without wishing to assert that the following explanation is the best, I conceive the mechanism of its production to be as follows: the increase of intrapulmonic pressure associated with ordinary and forced respiration is not sufficient for the ventilation of these collapsed structures. Cough, however, increases the intrapulmonic pressure at least fifty-fold above that which obtains during quiet or forced breathing. This sudden and decided increase of intrapulmonic pressure balloons out the collapsed areas and the rale is produced by the vibration of their contained secretion. The procedure should never be employed where there is a history of recent hemoptysis, or in conditions where an increased intrathoracic pressure produces pain: pleurisy, intercostal neuralgia, acute abdominal conditions, etc.

To summarize briefly, I would like to say that the value of certain of the traditional signs in early tuberculosis is open to question, and their repeated emphasis in diagnosis explains much of the confusion surrounding this subject. To my mind, the only helpful sign in early diagnosis is the *rale*, an extraneous sound. The diagnostic significance of the rale is greater than that of all the other signs combined, and, fortunately, the technique for its elicitation is simple and can be acquired after a few trials.

THE TREATMENT OF PNEUMONIA*

HARLOW BROOKS, M.D.

I HAVE very few remaining illusions about the treatment of pneumonia. Several times in my life I have thought I knew how to treat the disease, only to learn the very next week, perhaps, that I did not know as much about it as I had thought and that success in treatment often meant lowered virulence of the disease or better resistance on the part of the patient, rather than skill on my part.

I do not think there is a disease, if we look upon the pneumonias as a "disease," which requires more therapeutic versatility than does pneumonia, for in more cases than otherwise we must here individualize and treat, not a disease, but a patient with a disease.

I shall not, therefore, try to classify the pneumonias from any particular standpoint. I think it is very difficult to classify pneumonia from a clinically useful standpoint, except in one instance, namely, Type I cases. Here, I believe, a definite classification should be made in the interests of the proper clinical management of the patient.

As already stated, I have very few illusions about the accuracy and efficiency of my treatment of this disease. I have had just as bad records as any others. I have sometimes for short periods, or in certain mild epidemics, had just as good records any others. The treatment of pneumonia has nowhere near been solved. I am certain that I have not solved it and I am still forced to fall back on the individual and symptomatic treatment of the patient.

PREVENTION OF PNEUMONIA

In the first place, I think we have recently paid altogether too little attention to the preventive treatment of pneumonia. I have been interested in looking over some of the older textbooks of medicine and I find they laid a good deal of

*This lecture was delivered on Dec. 7, 1923.

stress on the preventive treatment of pneumonia. I think you may take up a good many modern text-books without finding a single word on the preventive treatment of the disease, and yet I am convinced that in a considerable number of instances it can be prevented. I am not so sure about the abortive treatment of pneumonia, but I am certain about the preventive treatment of epidemics of pneumonia because I have seen prevention accomplished in large groups of vigorous adults, living in very compact social conditions, where the course of any disease could be very accurately and certainly observed. I think every one who served in the army must have been deeply impressed with the value of prophylactic medicine as it was carried out there. I am perfectly convinced, as others no doubt were at that time, that you can, in large bodies of men, prevent pneumonia. If this can be accomplished in large groups of men, then why not in individuals?

I think the most important single thing to do in a pneumonia epidemic, either for the group or individual, when such a condition confronts us, is to give rest, as much as possible. Although pneumonia is, as we all recognize, a disease which attacks the vigorous and healthy to a very considerable degree—not the weakened ones—people who are acutally tired and exhausted are those most likely to contract the disease. This was very certainly shown by a study of the prophylactic treatment of the disease in the war.

Another old-fashioned idea in the preventive treatment of pneumonia, which has been largely relegated to the dim and distant past, but which I think should be resurrected, is the question of proper clothing in the prevention of pneumonia—adequate, proper clothing. That does not mean horse blankets or fur clothes in summer, but correct and proper clothing considering the time of the year, the character of the climate and the work which the man must do. These I believe are very important factors. To illustrate: A division which had been in France two months and had become thoroughly acclimated was living under what might be called ideal conditions for the army. They were quartered in well-ventilated, proper barracks, they were getting an adequate amount of food and rest. In brief,

everything was ideal with this division and the sick rate was very low. It was deemed advisable to send them hurriedly toward the front, not to engage immediately in actual combat, but to act as reserves. In the haste it was thought wise by an injudicious commander to discard all extra clothing, and the men went forward with exactly what they had on—no extra shoes or underclothes, but with overcoats, no ponchos, and carrying but a single blanket each. The weather conditions at that time were bad—rainy, cold and snowy weather. They traveled one night and the next day on fairly comfortable French troop trains. They were sent to a fairly comfortable camp ground, but it was raining and snowing, as it always was at that time of the year in France, in my experience. No tentage or other adequate shelter was obtainable. Inside of forty-eight hours we had nearly 10 per mille of pneumonia from that division, while divisions in combat in the same area gave us but a normal occurrence. I believe you agree with me that this could have been prevented.

Adequate food is another condition which should be considered in the prevention of pneumonia when dealing with large bodies of men. The same must apply to individuals. Poorly or inadequately fed men will contract the disease with much greater rapidity and with a much higher percentage of fatality than men who are well and properly fed.

Heat is another important thing which may be used to prevent pneumonia. It was found that if large quantities of hot soup, tea, or anything that was hot and nutritious, could be supplied to the men under great fatigue and exposure, those regiments had an immediate relative drop in the percentage of pneumonia occurrence. The more intelligently supervised British and French troops were supplied with alcohol for this purpose. Old medical authorities generally advised the same for exposed and exhausted individuals with similar benefit.

Early hospitalization is, I think, another important thing in the prevention of pneumonia. If a man has a catarrhal bronchitis or a cold, all of us know enough to put him to bed, unless he is a doctor, when he will probably refuse such a sensible procedure. Cases that are hospitalized early, that

is, put to bed, develop a small percentage of pneumonia following all the catarrhal infections. Those cases which are allowed to be up and about always develop a much higher percentage of pneumonia.

Unaccustomed exposure is a very important factor. That was evident everywhere during the war. A small body of troops was mobilized near New Rochelle. They were to be sent to Camp Upton. They were out one night marching and trying to seek shelter. It was not a particularly bad night—no worse than the nights on which many of us have to be out frequently—but the men of this new group, with their various infections, were thrown closely together and subjected to unwonted exposure. They had the agent of infection plus the exposure, to which they were not accustomed. Fifteen per cent of that group of men developed pneumonia.

DANGER OF TRANSMISSION OF PNEUMONIA

In civil life I have rarely seen direct transmission of pneumonia infection. I have never felt that a pneumonia case was a source of danger to the welfare of other patients in the ward. It is different with large groups of men in close contact with one another, such as large agricultural groups, industrial groups, and notably, soldiers. We know that it is altogether different under these circumstances, and that in such instances direct transmission of pneumonia does take place, given appropriate sensitizing factors. For example: We had a very small incidence of pneumonia among the negro troops at Camp Upton. They were mostly a fine set of men drawn from New York City and its environs. About two companies of badly infected men, as far as acute respiratory diseases were concerned, was sent us from the south. Inside of ten days after that, our group of negro soldiers topped our list in the percentage of pneumonia cases. There is no question about it: transmission of infection is an important matter in the prevention of pneumonia. In so far as we handle the disease in our civil hospitals, however, I have never seen it thus illustrated. Neither have I seen it in military hospitals, but in military groups in camp or barracks it is a very common and very evident thing.

INDIVIDUAL TREATMENT OF PNEUMONIA

Now, to get away from the subject of the prophylactic treatment of pneumonia, in which, as you may have guessed, I am much interested, let us take up the individual treatment of the disease.

I will confess at the outset that, to my mind, the most important thing in the treatment of pneumonia is to individualize. I do not think one can treat pneumonia by the hospital, or by the service, or by the ward; but it should and must be treated, if successfully, by the individual. It is the study of the individual case which determines whether your treatment is effective or not.

The Posture of the Patient. We have already stated that in the prophylactic treatment of the disease, early hospitalization is an important thing. I think that most of us will agree, that if we can take a case of tentative pneumonia, bronchitis, or catarrhal affection of any sort, and hospitalize it early —that is, put it to bed—we will in a very considerable percentage of cases at least avoid pneumonia. But, the question is, how shall we put them to bed? In the first place, a good many of the cases which are brought to your service, and to which you are called in private practice, have the disease fully developed when you first see them. If you ask some of them to lie down flat in bed, or force them to do so, it may add a great deal to their discomfort. I believe they should assume the posture which is most comfortable for them. A large number of cases, particularly those patients with large abdomens, from obesity, pregnancy, ascites or other conditions, find it very difficult to lie flat in bed. In such cases great relief is afforded by raising the back of the bed, or putting them on a bed rest, or even putting the bed on an incline. Some patients are very comfortable and breathe easier lying on the affected side, when only one side is affected. Others are more comfortable and breathe easier and are more quiet if they lie on the unaffected side, and so on. I believe, then, the posture of the patient in bed should be largely determined by his own desire and his own knowledge of comfort. Of course, one cannot allow them to sit up in bed and expose themselves unnecessarily when they are covered with sweat (although I have little fear of a patient running a pneumonia catching cold,

whatever we mean by that), but within reason patients do much better, in my experience, by adopting the posture in bed that is most comfortable for them. Of course, in hypostatic cases we should change the position of the patient very frequently. I believe that this should be adopted widely in the handling of ordinary cases of pneumonia. I believe that if you change the position frequently, within reason, of course, you lessen to a certain extent the tendency to the spread of the disease in the infected lung tissue. I think patients should be turned on one side perhaps for a few hours and then turned on the opposite side and then on the back and so on; but they must always be assisted in these movements and all unnecessary activities must be eliminated. Occasionally you will find a case which is most comfortable when flat on the stomach; and sometimes the patient is more comfortable with the head over the edge of the bed.

To recapitulate: comfort and rest are factors of primary importance in pneumonia. Patients can determine for themselves much better than we can the position most conducive to rest and comfort. This should be permitted within a reasonable degree at least. I believe we minimize the tendency towards hypostasis by changing the position of the patient at frequent intervals.

OPEN-AIR TREATMENT OF PNEUMONIA

I think this is something that must be determined by the individual case. At one time I had the opportunity of carefully studying this subject of open air as opposed to the closed-room treatment of pneumonia. The way I finally came to handle these cases was by permitting the patient to take that place which seemed best suited to him as an individual. In other words, I began by putting them outdoors at first. We had very comfortable sheltered porches. Patients put outside were well covered up. If they were cyanotic and became less so out of doors, if the temperature dropped, if they were delirious and became less so, or if the delirium cleared up entirely, we left them out of doors. If, on the other hand, when they were put out of doors, they became gray, cyanosis increased and dyspnea increased, back they went to the ward. A considerable number of cases did do better

when put back into a closed, warmed room. In other words, I believe the treatment in this respect depends entirely on the individual cases.

I think when I get pneumonia, I shall want to be put out of doors. I may be mistaken, but many cases, particularly those accustomed to being outdoors, do better outdoors than elsewhere, and the discomfort of the disease is much reduced in many cases. Patients from the tropics, I warn you, do badly outdoors. I have talked this question over with men who have practiced for a long time in the tropics. I have tried the out door treatment on patients from the tropics, from Cuba, Mexico, Porto Rico, Panama, etc.; and I think my colleagues from these districts are right in the opinion that such patients do badly under outdoor treatment.

Furthermore, I am convinced that exhaustion cases in elderly people, those who are debilitated by other diseases, such as typhoid, carcinosis, diabetes, or nephritis, generally the people who develop pneumonia as a complication after a severe surgical operation, anemic cases, and so on, as a rule, do badly out of doors. I believe they do much better indoors, oftentimes in a ward with a degree of heat that is uncomfortable for a well man. This is heresy, I recognize, but I am pretty sure it is a heresy which a good many of you have practiced. It is perfectly true, I am convinced, in scarlet fever, pneumonia and measles. At least, we found it to be so in our work at Camp Upton. Outdoor treatment in cases of pneumonia that developed as a consequence of one of these infections, ordinarily was not successful, whereas with indoor treatment especially in well-warmed wards, the patient did much better. This is particularly true in the catarrhal types of influenza. We had an interesting experiment of this kind quite involuntarily in a military hospital near Revigny in France. A division came out of the line very much exhausted from heavy combat and almost constant exposure to gas. They made camp near this little hospital of normally 500 beds. We got the message that this division was coming through with a lot of pneumonia cases. We already had 3500 people in our 500-bed hospital that night. It was raining and the men who were not sheltered in our hospital had to sleep on the

ground, and very little tentage was obtainable. So we had the divisional medical officers select the worst cases that they had. They turned over to us that night about 300 cases of tentative and actual pneumonia. For reasons of obligation, and not because we did the experiment voluntarily, we were able to put 150 patients into well-heated, in fact, overheated barracks and store rooms—poorly ventilated, but heated. We had some large stoves in these shacks and they were almost red-hot all night. The 150 cases remaining were put into similar rain-proof buildings which were just as good, or just as bad, as you might say, with the same amount of bed clothing, and the same abundant hot food, but there was no heat in these wards. Suffice it to say, inside of three days the cases that were in the overheated wards had dropped down to a very small number; that is, the number of cases which actually developed and the number of deaths which actually occurred were very much smaller than in the unheated, cold and equally poorly ventilated wards. Both groups received the same food and also the same attention except in the one respect of heat. I have no doubt at all but that chilled and exhausted men do much better with heat than with the lack of it in the treatment and prevention of pneumonia. Ventilation may be a very secondary matter.

DIETARY TREATMENT

This depends very much, in my opinion, on the status of the case. I ordinarily prefer to give my pneumonia cases as much fluid as they will comfortably take. If, on the other hand, they become quickly edematous, as some of them will, it is silly to give them more fluid. They should certainly be given less fluid under such circumstances. Some men treat pneumonia by depletion—the administration of calomel, salines, sometimes by venesection, the reduction of fluid, sweating, etc. These, in my experience, are sometimes advisable; again, it is an individual matter. I prefer to give my patients, who do well on it, a large quantity of water. If they fail to do well, if they develop edema, if the heart action is weak and grows worse instead of better, I cut off my fluids, perhaps entirely for a time. Milk and milk foods are my staples as far as diet is concerned. Soups and broths are

oftentimes beneficial, especially when you want to administer internal heat, as many cases require. They may have little or no caloric value as far as the food itself is concerned. Pneumonia is ordinarily a short-lived disease, and generally cases do better on less rather than on more calories. There are cases, however, quite the opposite, such as the anemic and the exhausted, which should receive, and obviously will receive, calories in amount sufficient for their requirements; for we are not treating the individual as we would like to treat the disease. Our pneumonia case is not a case of pneumonia, but a man who has unfortunately got pneumonia, and we must use common sense. In other words, I believe that we should give fluids where they are indicated, and dry diet wherever that may be indicated—in this case, few calories, and in that, all the food that digestion will tolerate.

With high blood pressure and edema present, no one thinks of giving salt. A possible exception may be Dr. Quimby, who was an enthusiastic believer in the specific benefit of NaCl in pneumonia, and who insisted that the salt treatment was indicated even in high tension and edematous cases.

Fruit juices, I think, are oftentimes among the best foods we can give. They are very grateful to the patient, and one can give a sufficient caloric value by the sweetening added to them.

I use alcohol in pneumonia principally as a readily utilized food. I think pretty nearly every other clinician does; also I resent bitterly some one in Washington or Nebraska telling me how much or how little alcohol I may use in any individual case. It is often one of the best things to be had in the treatment of cases of pneumonia, particularly so with those persons accustomed to it as an article of regular diet, as are the French and Italians, etc. I use it also particularly in those cases in which exhaustion is a very marked symptom. These, I think, do exceedingly well when alcohol is administered, not as a medicine, but as a food.

Alkaline waters I use quite extensively. I believe I am using them more and more as I go on, because I believe acidosis is present in a very high percentage of cases of pneu-

monia. I think this acidosis is relieved by the use of alkalies, if given in the form of water—Seltzer, Vichy, any of the alkaline waters. With them we have at least an easy way of administering the alkali.

GASTROINTESTINAL TREATMENT

I believe that one of the essential things in the early treatment of a case of pneumonia is to clear out the intestinal tract; and that is, of course, most effectively done by the use of purgation of some sort. I do not use violent purges, except in cases in which depletion appears to be a desirable thing, and there are such cases. A powerful, well- or overnourished patient is better off because of it. I believe that when such cases enter the hospital, they are improved by a good cleaning out; but the little, weak, skinny fellow on the other side of the ward would not do so well with that sort of thing. Laxatives should be used in the latter cases, preferably the salines. High colonic irrigations are very satisfactory in cleaning out the colon. We ordinarily use the alkaline solution, sometimes 5 per cent bicarbonate, sometimes 10 per cent, sometimes salts other than bicarbonate of soda—sugar solutions and the like—both for cleansing purposes and also because a certain amount of fluid and drug is thereby absorbed. Of course, there are cases in which one would not dare to use laxatives, cases in which there is a very narrow margin of reserve in so far as the heart action is concerned, cases which are already more or less in extremis and in which rest and absolute quiet appear to be most desirable factors.

Sugar solutions I use in a good many of my cases, especially those exhausted and tired out from pre-existing or intercurrent disease which may have preceded or succeeded the infection of pneumonia. It is a very nice way to administer food value, because there is a considerable amount of sugar taken up when it is administered in the proper way by the colon.

TREATMENT OF TYMPANITES

This I regard as one of the most difficult and most annoying conditions arising in the treatment of pneumonia. I have had better success in the treatment of tympanites by the use of

colonic lavage than from anything else, but there are cases, and a good many of them, where this is neither indicated, nor of value. I saw one this afternoon in which we did nothing beyond applying heat to the abdomen. In those cases in which lavage, with thorough cleaning out of the intestinal tract, does no good, one must resort to other measures. Sometimes very simple drugs will relieve the condition. The old-fashioned essence of peppermint or aromatic spirits of ammonia not infrequently will give relief. I have never had any benefit from the use of musk, though it is highly recommended by some of my friends.

Pituitrin, I think, is a legitimate drug to use here. If you use it carelessly you may meet with catastrophe; at least, in several cases I think I met with calamity as a result of the unwise use of the drug. However, there are cases in which an ampoule of pituitrin relieves the condition of tympanites most promptly. Caffein sometimes does the same thing. Adrenalin, especially when the blood pressure is very low and the patient in an asthenic condition, and when the tympanites appears to be due to a paralysis of the gut, is sometimes of value. Strychnin will occasionally do the same thing. Sometimes the application of heat will give complete relief. Sometimes the ice bag will give relief. We must experiment, trying first one thing and then another. Gastric lavage will oftentimes give relief, but of course, one is a little careful about attempting that, in patients who have never had their stomachs washed out. It is a rather disagreeable procedure on the first occasion, but oftentimes it gives great relief, especially if bile is regurgitating from the duodenum into the stomach. I only wish I knew something that would *always* relieve my patients of tympanites. There is no symptom, except cardiac failure, that I dread more in pneumonia.

THE PHYSICAL TREATMENT—COUNTER-IRRITATION

A great many people still use counter-irritation in the treatment of pneumonia. I have not done so for a good many years, and my reason is because of the fact that I could never see at autopsy on these cases that I had accomplished anything except to make the patient more uncomfortable. I

have blistered the skin and used the actual cautery. I have made the patients much more uncomfortable undoubtedly, but I never did anything that caused any apparent relief inside the thoracic wall. I do not disagree with the man who uses blisters, hot mustard plasters, or anything of that sort, if he feels he makes his patient better thereby; but I am too much of a skeptic to see how it effects any relief. I understand the theory of the effect on the peripheral nerves and all that sort of thing, but I never saw at autopsy, in cases that had received this sort of treatment, any changes in the lung which led me to have any faith in it whatsoever. It usually adds to the discomfort of the patient.

HYDROTHERAPY

Before condemning hydrotherapy, as I am going to do, I will tell you something about my experience with it. I am not a heretic born nor a heretic bred, and I worked for some time with Dr. Baruch; for I had the privilege of having him take charge for a time of a ward of my pneumonia cases at Camp Upton. He brought down his own nurses, and as far as possible we did everything exactly as he wished it done. He stayed there with us for some time and devoted himself in a perfectly wonderful way in trying to convert us and improve our patients. He did not convert us at all. I think even his own faith was a little bit shaken by comparison of the cases that received hydrotherapy with those that received only the ordinary symptomatic treatment. I believe that the cold sponge will sometimes relieve the symptoms of the case, and oftentimes lower the temperature and give a good deal of relief; but otherwise hydrotherapy has made my patients worse instead of better, because it exhausted them. I believe that the less you disturb pneumonia cases, the less you turn them over, the less physical excitement of any kind you cause them, the better chance they have of getting well. Even when given under the expert hands of Dr. Baruch, a considerable amount of unrest, mental and physical, resulted from this treatment. A simple sponging and cleansing bath, which should be given as quietly and with as little disturbance as possible, makes the patient a little more comfortable and does him good; but

beyond that I see nothing in hydrotherapy which makes me feel hopeful about it as a measure in the treatment of pneumonia.

The most important thing you can do for a pneumonia patient is to give him just as much rest as possible. Let him be. Don't be too curious about him. Don't be too scientific and try to find out whether the spot has gone 2 cm. to the left of where it was yesterday, and all that sort of thing. Don't let him sit up in bed; and I am sure he will be grateful for a certain type of absent treatment. Doctors I have treated tell me oftentimes: "I am so glad you did not move me around or try to make an exact anatomical diagnosis."

VENESECTION

I believe that venesection is a very useful method of treatment, particularly in plethoric cases. Great big longshoremen, iron workers and piano movers, such as we sometimes have in the City Hospital, are ordinarily better for a vigorous blood letting. I think also, particularly in the early stage of the disease, if the blood pressure is high, cyanosis marked, the veins full and the heart action labored, you will improve that case by venesection. I would take away enough so that my patient was breathing a little easier. If he did not breathe a little easier after removing 300 or 400 c.c. I would stop. I would probably stop before that. If he breathes easier as we proceed I might take away a little more until the dyspnea is relieved.

I do not use venesection in the period of convalescence, even if the heart is going wrong, because I believe one thereby depletes the patient and lowers his resistance against the disease.

THE TREATMENT OF FEVER

I do not treat the fever very much. Those cases acceptable for outdoor treatment will oftentimes show a beautiful drop in temperature when put out of doors. Oftentimes a cold sponge relieves the temperature very much. Sometimes lavage with cool or even cold water reduces the temperature and makes the patient more comfortable. It sometimes,

and sometimes not, relieves the delirium which is likely to develop in certain cases of hyperexia.

I have tried and given up the drug treatment of fever. I will no doubt try it again as new drugs come out. However, no drug has as yet done me a good turn in the reduction of fever.

THE TREATMENT OF PAIN

I have already said that I believe in giving the patient rest. I regard it as the most important thing of all in the treatment of pneumonia. Sometimes a patient has very great pain, perhaps from the pleurisy, or pericarditis, or perhaps from an unexplained condition. Headache sometimes develops even without a meningeal involvement, and may be the cause of pain. Sometimes a true neuralgia, a neuritis, apparently develops in these pneumonia cases. If he has pain and cannot rest, I believe in relieving the pain. I give anything that I can find. Opium—I realize the reasons why one may not theoretically give opium, or should not give opium in certain cases of pneumonia; but I believe the giving of rest is much more important than any of those reasons, which are largely theoretic. My patients do better when they receive morphin, codein, heroin, etc., as may be indicated, to relieve them of the sharp edge of pain which spoils the possibility of sleep and keeps them in constant agony and unrest, both mental and physical. No harm, I believe, is done by any of the opium group in this class of cases. One must be cautious about using other sedatives, some of which I am afraid of. They should not be used unnecessarily when the blood pressure is low. I rely mostly on the opium group for relief. I will return to that again a little later, when we are discussing the question of cough.

In reference to heroin, I was asked today if I didn't think we should give it up. I don't think I prescribe it over five or six times a year, but when I do prescribe it, I use it because my other forms of hypnotics have failed. In not a few cases heroin will do what no other drug will do. Every man who has practiced medicine widely—not worked in an experimental pharmacological laboratory only—will, I am sure, endorse my statement that each drug has its merits and patients oftentimes refuse to comply with guinea-pig conclusions. Some-

times heroin will produce an effect that none of the other drugs will produce, I hesitate to tear any good string from my small therapeutic harp.

THE TREATMENT OF COUGH

If you watch a pneumonia patient cough, you will observe that after the paroxysm of coughing he will usually draw a deep inspiration: he will draw in his breath in order to reinflate the lungs. When he coughs, he throws into the larger bronchi a large amount of infective material from the involved area of the lung. When he aspirates he pulls that back somewhere, perhaps into healthy areas; and I am certain that the persistence of cough tends to the spread of the infection throughout the lungs. I believe for that reason that cough should be controlled, if possible. By this I mean the giving of morphin, heroin, aspirin sometimes, codein, etc., to the point of giving relief. Sometimes, to stop the cough, I have given inhalations of chloroform. Cough is very exhausting, it is a great strain on the heart, and it is a symptom which I believe spreads the infection and tremendously exhausts the patient. I think it should be controlled as far as possible.

THE TREATMENT OF CYANOSIS

This brings up the question of the oxygen treatment in cases of pneumonia. I date back to the period when every bad case of pneumonia got the "blue can" and the use of oxygen was universally indulged in; so I am not a fair jury to try oxygen. Oxygen will give relief in some cases of cyanosis. It occasionally, much more rarely, will relieve the dyspnea of pneumonia. If it does relieve the dyspnea, let us have oxygen. If it does not, let us not use it. If it relieves the cyanosis, I have no objection to it, unless it is given too often, or causes a drying of the mucous membranes. The newer methods of the administration of oxygen have been widely admired by a great many physicians. Dr. Tasker Howard, for whom I have great respect and admiration, feels very favorably inclined toward the use or administration of oxygen by some of the newer methods; that is, the mask over the face. I saw 2 cases in which he showed to

my satisfaction that he was improving the patients by the use of oxygen. In my general experience, in a large number of cases, I have felt that the annoyance caused the patient was very inadequately paid for by the theoretical benefit which they may have received from the use of oxygen. An abundance of fresh air, and, if contraindications do not exist, cool air, is very much more desirable, to my mind. I confess, however, that there are cases, few in number, in which the administration of oxygen gives symptomatic benefit of very real and definite value. I believe that in any case that does not show almost immediate relief from the use of oxygen one is wasting the patient's energy in forcing him to take the drug in that way; but if it gives relief, even though it be but symptomatic in any instance, let us use it.

THE SPECIFIC TREATMENT

This is something which I must pass over very rapidly. Dr. Cecil prophylactically vaccinated over 15,000 of the 77th Division against pneumonia, before they went to France, using all the standard strains. We got permission from the Surgeon-General to send a young man over there to observe those men. The last time I heard from that young man he was doing kitchen police duty. Nobody knows what happened to those men who got a protective vaccination from pneumonia. I have tried it on the outside, as no doubt many others have done. I am still open to conviction. I have seen cases in which I felt that the prophylactic treatment of pneumonia by vaccine was a legitimate method. I am not prepared to say today whether or not it is sufficiently universal to justify our using that method to any considerable extent.

THE VACCINE TREATMENT OF THE DEVELOPED CASE

Last winter, with Dr. Danzer, we used this treatment in practically all our cases at City Hospital, Blackwell's Island. The interns were quite enthusiastic over the treatment. I think that Dr. Danzer and I were much more cool about it. We were willing to be convinced, but I am not convinced yet, and I do not believe he was, that we really

did those patients any more good than was explained by the so-called symptomatic treatment. Dr. Cecil is getting some interesting results from his polyvalent serum, which is practically universal. I have tried to follow his work, but I am not yet convinced that he is getting anything in the way of definite results. However, I am most hopeful of work along such lines by men of such wide laboratory and clinical experience as Dr. Cecil.

THE TREATMENT OF TYPE I CASES WITH TYPE I SERUM

I have no doubt whatever that the use of Type I serum in Type I cases, if given early, within the first forty-eight hours, is a splendid thing. It is, in my opinion, comparable to the use of diphtheria antitoxine, but it should be used early or not at all.

My experience with a large number of cases makes me feel perfectly sure, however, that if you give Type I serum to Type II, III, IV, or mixed infections of any kind, such as influenza cases, you lessen the likelihood of those patients getting well. You introduce a foreign serum which does more harm than good. It is only in Type I cases that it is good. If it is given early, it is excellent. If serum is given late, it causes more disturbance, I think, than good.

Other Foreign Sera. Joseph Miller and Joseph Capps, two of my colleagues in Chicago, have used chicken serum and other foreign serum with equally good results, as they believe, in Type I cases as with Type I serum. I cannot agree with them on that point. I have seen cases receive foreign serum, such as chicken serum or horse serum, which seemed to show a beneficial reaction after administration of the serum treatment, but there has not been a sufficient number to make me feel that the serum has a general application. Possibly I have not seen enough cases.

Dr. Solis Cohen's quinin treatment, I think, has a basis, as far as it goes theoretically. He, I think, however, obtains his fine results largely through the careful individual attention which he also gives his cases. I value the personal time and effort which he gives to them more than his quinin treatment. However, there is a good theoretical basis for the use of quinin, and I have certainly no quarrel with the man

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who uses it. I do not get the results Dr. Cohen gets, however, with the method.

THE IODIDE TREATMENT

I use the iodide treatment in cases which are not resorbing properly, not convalescing properly, but I find no effect whatever with it in acute cases of pneumonia, except in lues, where I believe it to be very valuable.

The use of calomel, salines, etc., is a legitimate method in the type of cases that are usually venesected. In the dry cases one gives it up. The treatment of Quimby—the salt treatment—I can only mention to condemn it. I have found it does more harm than good.

DIGITALIS TREATMENT

I don't like to use such a term, for I don't think it is a treatment for pneumonia. However, I believe that if I had just one drug to treat *cases* of pneumonia, that drug would be digitalis; and I think that would be the verdict of many others. If you had just one drug to use in pneumonia cases, you would probably choose digitalis. The reason for that is that most cases of pneumonia, when they die, die of cardiac failure. I analyzed 5000 cases of death from pneumonia in France, and in nearly all of them the preponderating cause, the immediate cause of death, was circulatory failure. I don't give digitalis unless I feel it is indicated in apprehending cardiac failure or in coping with cardiac incompetency. I do not give digitalis to every case of pneumonia. I confess I give it to most cases of pneumonia, because in most cases in adults (I see children only very rarely) symptoms or evidences of cardiac incompetency eventually develop. Some cases do better with strophanthin. These are mostly those ill-defined cases which fail to respond to digitalis, but do respond to strophanthin. I cannot detect them except by experiment.

Convallaria, which contracts the right heart more than the left, has been recommended by Dr. Lambert and Dr. Delafield. I believe that it has done me good service in several instances.

I am sorry to be obliged to pass rapidly over this subject of the use of the digitalis group of drugs, about which I am very enthusiastic.

Camphor and caffein I use a great deal. I use camphor for an effect I hope to get in a few instants and for the effect which I expect will pass away in thirty or forty minutes. I am not afraid of giving large doses. Sometimes I believe camphor has tided over a patient and given me a recovery where without it I should have lost the patient. The same is true of caffein. It is a splendid drug, particularly where the renal action is defective and toxic conditions are prominent. Oftentimes I have given it with the alkalies. I think it is very effective.

Strychnin is very old-fashioned in the treatment of pneumonia. I believe sometimes it helps, particularly when it is given with digitalis, because it makes the heart muscle more irritable and preserves longer, I believe, the irritable impulse which lies at the base of contractility of the heart muscle.

TREATMENT OF MANIA

The treatment of the mania that develops in pneumonia and oftentimes after pneumonia, is very interesting. It is a difficult problem, especially the treatment of the so-called convalescent type of mania. In the treatment of this symptom, as far as drugs are concerned, I have had the best success by giving bromides. Luminal is spoken of very highly by men who have had a large experience with it.

Chloral hydrate is a splendid drug to control the restlessness and mania which develop so frequently after pneumonia and sometimes in the course of it, and I see no reason why it should not be used. We do not like to use it in the presence of a low blood pressure.

The question of convalescent treatment I shall not attempt to discuss.

SUMMARY

In concluding, I will repeat very briefly some important points regarding the treatment of pneumonia:

In the first place, give your patient rest, all the rest that you possibly can.

Except in Type I cases, the treatment is essentially symptomatic, and symptomatic treatment not of a disease, but of an individual with a disease.

I am perfectly convinced that whether or not a pneumonia case survives is not so much dependent on the type of treatment as on the persistence, patience and individual attention given by the physician.

I am particularly sorry that I have had so little space for the consideration of the use of the digitalis group of drugs in pneumonia, as I lay great stress on its value in most unfavorable cases, but my time is gone, and I fear your patience with it.

APPENDICITIS*

WILLIAM FRANCIS CAMPBELL, M.D.

WHY? is the master word in human progress. When man began to ask the question Why? and got curious about things, the anterior portion of his spine began to assume a bulbous shape, and it has continued to increase in size ever since.

We all find ourselves every once in a while going off at a tangent, seeking false gods, getting off on side tracks. If we are to maintain a sound and sane habit of mind we must get back every now and again to fundamentals. This is our aim in the present discussion.

I have in my library a book of a little over two hundred pages, written by an Englishman some sixty years ago. If I had the choice of just one book in my medical library I would select this one. It deals with two fundamentals; and I am sure that if I stick to those fundamentals I shall never go very far astray in the practice of medicine. The name of the book is "Hilton's Rest and Pain."

Pain is the master symptom of disease, and rest is the master remedy; and Hilton shows the relationship between pain and rest. He demonstrates that pain always produces a condition of the muscular system that brings about quiescence and rest, in order that this therapeutic measure may be applied. He shows how, on an anatomical basis, this is brought about. For instance, if you have an inflammation of the shoulder-joint, the first thing you will notice is that you cannot and do not want to move it. What has happened? The pain has irritated the nerves that supply the muscles; the muscles around the shoulder-joint are put in a state of spasm, and this state of spasm produces a splinting of the shoulder-joint so that you cannot move the joint. This is because "the nerves that supply the joint supply the muscles that move the

*This lecture was delivered on Nov. 24, 1922.

joint and the skin over the muscles." This is one of Hilton's laws.

The abdominal cavity is nothing but a joint, a large joint, with movable viscera, lined, just as is other joints, with a serosa that has a lubricating fluid. The same thing applies to the abdominal cavity that applies to the shoulder-joint, namely, that with an acute inflammation of the abdominal cavity there is immediately a splinting of the abdominal muscles which give us what is known as rigidity. It is nothing more than a spasm of these muscles, an effort on the part of nature to put the contents of the abdomen in a state of rest.

Pain is the master symptom of disease. Pain is fundamental: it is the raison d'etre of our profession. Yet there are few who can give a definition of pain—a rational definition, one that is satisfactory, one that meets the indication. If one feels that pain is just something that causes humans to suffer and is for the sole purpose of alleviation, one's conception of pain is very circumscribed. You must have a bigger conception of it in order to treat pain properly. Pain is not the enemy of humanity: it is our friend. It is not a curse: it is a boon to humanity. It is our protector. If anybody should endow me with a supermagic by which I could abolish pain from the earth I would refuse to take the commission; for the human race cannot exist without pain: it would disintegrate.

Supposing we never had a toothache; what would happen? The teeth would become decayed and fall out. No one would go to a dentist. There is no inconvenience, there is no pain; what is the use? The teeth would simply disintegrate and disappear.

Supposing there was no pain with appendicitis. There would be no reason for anyone's going to the doctor. There is just an inflammation, ulceration, perforation, peritonitis and finally death, because the patient was not urged in some way or other to take care of himself and call in assistance.

Now, pain is not only the alarm that notifies us that the machine is out of order, but it is the *physiological urge* which simply makes us do something to relieve the pain, and will not let us alone or give us any peace until the damage is

repaired. Hence, *our first duty in reference to pain is not its alleviation, but its interpretation.*

What does pain tell us? What tissue is crying for help? After we have interpreted the pain, then it is time to remove the cause and alleviate the pain.

Now, what has this got to do with appendicitis? We have been studying appendicitis intensively for the last thirty-five years, and we in Brooklyn have a tradition to live up to. There was once a great surgeon who lived in Brooklyn; it was not so many years ago, and some of us had the pleasure of working under his inspiring leadership. That surgeon made one of the finest contributions to the problem of appendicitis that has been made by any surgeon in the United States. I refer to the late lamented Dr. George Ryerson Fowler, after whom the present "Fowler position" is named.

Now, where do we stand concerning appendicitis at the present time? I want to cite a few statistics that may give us food for serious thought.

I will call attention, in the first place, to the death rate of some common diseases per 100,000 of population in 1910 and 1920. We find that in pulmonary tuberculosis 178 died in 1910 and 154 in 1920. We made some progress there. The death rate is less in 1920 than it was in 1910. Organic heart, 137 in 1910 and 128 in 1920. We made some progress there. In pneumonia 136 in 1910 and 110 in 1920. We made a good deal of progress there. Bright's disease, 119 in 1910 and 105 in 1920; also some progress.

Now, what has happened in appendicitis? We find the figures for 1910 were 16.1 and in 1920 26.4, a gain of 10 on the wrong side in ten years. Now, isn't that food for thought? What is the trouble? Why cannot we show as good, if not better, statistics for appendicitis than for these other diseases, especially when we are dealing so familiarly with it every day?

Now, another point; and this is also food for thought. The mortality of appendicitis in hospitals with well-organized staffs is 2 per cent. I refer to hospitals that have a regular attending staff, thoroughly organized, and kept up to date. In hospitals with unorganized staffs—that is, where

anybody can operate who brings a case—the percentage is 8 per cent.

Now, somebody is taking too long finding the appendix. Somebody is fumbling in the abdominal cavity, or the statistics could not be 8 per cent as compared with 2 per cent.

I want to call your attention to the fact that 40 *per cent of the cases that the surgeon sees are late cases.* Now, who is at fault? I don't want to put all this blame on the family doctor; it is not all chargeable to him.

There are three factors in this blunder. The physician is one, the patient is another; and then we come to that indefatigable, ever-present, hypercritical, over-emotional "Relative." You can deal with the patient; there is no trouble about that. But it is this "Relative" that hangs on your coat-tail and walks out with you to the door; and you explain again, after explaining several times, the seriousness of the case and the fact that the patient ought to be kept very quiet. Then as you are about to go down the step this person calls you back and says: "Doctor, what can we give the patient to eat?"

I want to present you at this point with a "Vade Mecum." That is, "go with me," "be my companion," "be at my elbow so that when I need you I can use you." There are various methods of approaching this problem. I only present you with a method which I have found helpful to myself. I don't say it is the only method, or even a particularly good one. It is *a* method, and it happens to be my method and the one which I value more than any other.

In approaching this subject of appendicitis, the thing for us to do is to realize that *in order to reduce the mortality of appendicitis we must arrive at an earlier diagnosis and better treatment.*

Now, how can we get at earlier diagnosis? By means of the "Vade Mecum." In approaching a case of inflammation within the abdomen (appendicitis) I try to find out, in the first place, not what organ is involved, but whether there really is an inflammation present. That is the important thing to find out. Never mind whether it is due to the appendix, the gall-bladder, the pancreas or something else. The point is, is there an inflammation in the abdominal

cavity? Let us take a concrete case. We are called in to see a man with an abdominal pain which he has had for several hours. We will not begin by trying to discover whether it is the appendix, the gall-bladder or anything of the kind. The point to decide is, whether this man has an inflammation in his abdominal cavity, whatever organ may be the cause. Now, how will we know? We must find out whether he has an acute abdomen. An acute abdomen is a symptom-complex consisting of four symptoms: *Pain, vomiting, tenderness, rigidity*. Now, in going over this case we take our symptom-complex of the acute abdomen. Never mind what organ is affected. We must go over the abdomen and simply say: have I got the pain, the vomiting, the tenderness, the rigidity? If so, I have an acute abdomen. Make that diagnosis and be satisfied you have an acute abdomen. Next, apply the rules of the acute abdomen. You may find out later whether the inflammation is in the appendix, the gall-bladder, the pancreas or somewhere else.

This acute abdomen is surgical, and the treatment is surgical; so, having established an acute abdomen, we will make no mistake by simply treating it in a surgical way and applying "*the rules of the acute abdomen.*"

What are the rules of the acute abdomen? They are three in number:

Rule No. 1. Never give a dose of morphin until you have made a diagnosis and determined upon the line of treatment. Morphin before diagnosis is one of the worst things you can give, because it masks the symptoms. It puts the patient in a state of well-being. It is the "fool's paradise" that fosters that spirit of procrastination which has done more toward increasing the mortality of appendicitis than any other one factor. After diagnosis, morphin is indicated, because then you relieve the patient's distress; and the very best thing you can do is to give him a dose of morphin in preparation for the anesthetic for the operation.

Rule No. 2. *Close the mouth and keep it closed.* No food and no cathartic. *Nothing by mouth.* The thing we are trying to do is to bring about a condition of rest. Hilton's law of rest is what we need to remember. Rest is the therapeutic remedy. The one danger is peristalsis. And what causes peristalsis?

Food and cathartics; and the one thing to do is to give nothing by mouth whatsoever, not even water. Supply the water, if you choose, by means of a Murphy drip, or by means of small enemas. Give water by rectum every three to four hours, 6 oz. in quantity.

Rule No. 3. *Never give a cathartic in acute abdomen.* The purge is the most deadly medication we can give. Can you imagine anything worse, with a focus of infection in the coils of the intestine, than to give a cathartic and set up these waves of peristalsis? You might as well take your patient and churn him, or put him on horseback.

No cathartics, no food, morphin after diagnosis—those are the three rules of the acute abdomen.

Now, then, we have found that the patient has an acute abdomen because he has these four symptoms: pain, vomiting, tenderness and rigidity. To use a musical figure, these four symptoms are the four fundamental notes in the symphony of the acute abdomen on which the various viscera play their typical tunes. The viscera, when they are inflamed, take these four fundamental notes and play a tune of their own, which, if you can recognize, will enable you to make a diagnosis of the cause of the acute abdomen. I mean that appendicitis takes these four fundamental notes and varies one or two of them a little bit, but always in the same characteristic way; and it plays a tune of its own with those four fundamental notes. *A trained diagnostic ear recognizes the tune.*

Observe acute intestinal obstruction. It takes those four notes and varies one or two of them in a certain way, and it plays its own little tune. It is the same way with acute pancreatitis. It is the same way with acute cholecystitis.

All the way through, the acute abdomen is the foundation of diagnosis; and the interpretation of the variations in these four fundamental notes is the diagnosis of what particular viscus is involved.

In appendicitis, it is not necessary to go over the differentiating pathology; for chronic appendicitis, catarrhal appendicitis, ulcerative appendicitis, perforative appendicitis and gangrenous appendicitis are all the same pathological process, only at different stages—the same pathological process exactly: infection in a tube of small caliber, and

one that does not drain well. Different stages of the same disease.

Now, then, 80 per cent of all the intraabdominal lesions are appendicitis. Four out of every five abdominal pains are appendicitis. The other 20 per cent are divided between the pancreas, the stomach, the intestine and the female adnexa. So, even after a very cursory examination, if we diagnose appendicitis, we would be right four out of five times.

Appendicitis is not, as a rule, difficult to diagnose, as 98 per cent of the cases follow a regular symptomatology. It is not simulated by any other disease. That is encouraging, also.

What are the symptoms of appendicitis? They are the symptoms of the acute abdomen, but with the little variations that the appendix plays. In the first place, pain, sudden pain, right out of a clear sky. A man is in perfect health; he has probably eaten a hearty meal; he goes to bed and wakes up with a sudden, acute abdominal pain, colicky in character, and generally all over the abdomen.

We see how the appendix is varying this one note. It is just making it typical of itself. After a while this general pain localizes in the right iliac fossa. That is the variation which the appendix makes with the pain. Then we get vomiting, which is a transitory affair, and soon over. Then we have the tenderness. This is localized, in general, over the appendix, but not always in the right iliac fossa. There we must be careful. The cecum, or the appendix, isn't always in the right iliac fossa. True, it is in the majority of cases. The appendix is the tail of the cecum and, as a rule, the cecum lies in the right iliac fossa; consequently you get the appendix there. But in exceptional cases the appendix may lie anywhere in the abdominal cavity. How shall we account for the variations? To go back to embryology for a little: The cecum in fetal life rotates and migrates up from the left side around under the liver and down into the right iliac fossa, and there it finally lodges, if it is normal; but in a case of arrested development the cecum may settle anywhere, but it will go down to its usual place in the majority of cases. In some cases it will migrate into the pelvis, and then we have what is known as the "pelvic appendix." This is a very impor-

tant appendix, as Dr. Jennings brings out in his article, showing that 26 per cent of our appendices are pelvic ones. This means that when there is tenderness in a pelvic appendix we cannot feel it by pressing downward in the right iliac fossa; we must make a vaginal or a rectal examination. In cases of appendicitis, this is very important. I believe, also, that the majority of our left-sided appendix cases are pelvic appendices. At least, I have found it so.

The next symptom is rigidity of the abdominal muscles. This is always on the right side; the muscles are thrown into a state of spasm in an effort to protect the viscera beneath; and we have rigidity of the right rectus muscle or of the right external oblique.

We also have temperature, as a rule, of 102° or 103°F. This is not of much value. In some of the worst cases of appendicitis there is a normal or subnormal temperature. In gangrenous appendicitis, when the pain suddenly ceases, we find that the temperature goes down. The pulse, however, keeps very rapid, and we have a very serious condition with a normal temperature.

There is also a normal temperature in many cases of appendicial abscess.

The pulse is a reliable sign of irritation of the peritoneum. In cases of gangrenous appendicitis there is a very thready, rapid pulse. Notwithstanding the temperature, there is always an increase in the pulse rate wherever there is peritoneal irritation. Taken as a whole, I would rely more upon the pulse than upon the temperature in the diagnosis. In some cases of appendicial abscess, where the toxemia has been well walled off and is no longer being absorbed, we frequently have a normal pulse.

Leucocytosis—the blood count—is of no diagnostic value in appendicitis. It is simply indicative of an inflammatory reaction in the tissues of the body, and is not diagnostic of appendicits: it is simply corroborative.

Tumor is of value as a late symptom. When the appendix has ruptured, the contents of the appendix are thrown into the abdominal cavity, and the omentum, the abdominal policeman, wraps itself around it and strives to protect it from the rest of the cavity with exudate. With the exudate

splinting the coils of intestine you get an appendicial abscess; but the doctor who waits for a tumor to form in the abdominal cavity before making a diagnosis of appendicitis is guilty of malpractice.

Lameness in appendicitis is due to the appendix irritating, by its inflammatory products, the sheath of the psoas muscle: In consequence, the psoas is thrown into a condition of spasm and there is a little flexion of the thigh which causes the patient to walk lame. It is very significant of appendicial inflammation in relation to the psoas muscle, but it is a late symptom, and of no value in the early diagnosis of appendicitis.

Now, then, there is one thing I want to say about the diagnosis of appendicitis in children, which is very important. I believe the majority of abdominal pains in children are due to appendicitis. When called in to see a child with pain, temperature and vomiting, the first thing to think about is appendicitis, and the second thing is pneumonia. Always when called in to see a child with an abdominal pain, go over the lungs and be sure there is no pneumonia present before proceeding to make a diagnosis of "acute abdomen."

When we have a child to deal with we are not dealing with a miniature adult, by any means, but with something entirely sui generis. A child cannot analyze its sensations: It cannot interpret its feelings. It cannot cooperate with the doctor: How shall we make a diagnosis in a child? First, rely on the history, and second, on the objective symptoms which you can elicit. The history elicited from the parents will indicate that there has probably been some little chronic gastrointestinal disturbance. The objective symptoms obtainable from a child are fever, vomiting, distress, the fact that the patient is not comfortable; and not much else. It is impossible to tell whether a child is tender, or whether he has rigidity or not, when he is screaming and putting up such a muscular defense that he will hardly permit you to feel the abdomen at all. What shall we do? *Always make a rectal examination.* You will find your focus by this means. I think that by routinely making a rectal examination of all cases of appendicitis one gains much valuable knowledge and avoids many mistakes. One can always make a rectal examination on a

child; the examining finger can explore very readily the cecal region, because in children, the cecum is not far from the rectum.

Another thing which I have done in children, in order to make sure I have an "acute abdomen" to deal with, is to give a light ether anesthesia of two or three minutes so as to make sure just exactly what I had, before proceeding to operation.

Now, for a consideration of the treatment of appendicitis.

Having made the diagnosis of appendicitis, the treatment is surgical from the very start. There is no such thing as the medical treatment of appendicitis. Moreover, the family physician must get the same outlook as the surgeon if he is going to cooperate properly. As for the physician starting a line of treatment, and then, when he finds his medicaments of no avail, calling in a surgeon, that is not the treatment of appendicitis. There is no medicine in the pharmacopeia that has any effect upon the appendix. We were almost beginning to think antiphlogistin had, but familiarity has only bred contempt.

As to the place of the family physician in the treatment of appendicitis, he and the surgeon should work as a team. As I have said, they are not competitors; they are partners in this work of giving relief. The treatment of appendicitis to this extent is two-fold: first, preoperative; second, operative. The preoperative treatment is in the hands of the physician. He should place the patient in the best possible condition to receive the benefits of operation. The surgeon performs the operation. The physician is not going to throw up his hands and run away, saying, "This is a case for a surgeon." He is going to stay right on the job and do a specific work which is just as necessary as the surgeon's work. If he doesn't do that work well, the surgeon's work won't be of very much account.

The problem is to be handled as follows: The family physician makes a diagnosis of appendicitis. He knows the patient is to be operated on. He prescribes rest in bed—a difficult thing to secure. He preaches this thing, and then goes to the house and finds the patient sitting, dressed, on the side of the bed.

That is not rest in bed. Rest in bed with the Fowler position—that is the way to put the patient at rest. Not that I believe the Fowler position has any effect at all in relation to the slower absorption of toxins in the pelvic peritoneum. It has been proved anatomically that the pelvic peritoneum does not absorb the exudate any slower than the peritoneum of the diaphragm. I believe the Fowler position, however, is the anatomical, the biological position, the position of rest in which your patient will be most comfortable.

After placing the patient in bed, close his mouth, and keep it closed. No such thing as food or water. Give him a Murphy drip, or a small saline enema every three or four hours, and then a dose of morphin. The dose of morphin is given after the diagnosis, and as such it will put the patient in the best possible condition of quietude. It will be a real aid to him in taking the anesthetic later on. With the rest in bed, the Fowler position and the closed mouth, the patient, brought to operation, will receive its benefits in the best possible condition.

SURGICAL DIAGNOSIS*

JOHN B. DEAVER, M.D.
AND
STANLEY P. REIMANN, M.D.

IN my work in the Lankenau Hospital, Dr. Stanley P. Reimann, Chief of the Pathological Department, is in intimate touch with me and my patients at all times, and, I am glad to say, to the interests of both. Without this intimate association, I am sure my part of the work would not be as successful as I hope it is; I have therefore taken pleasure in associating him with me in the preparation of this address.

Surgical diagnosis comprises not only the diagnosis of the condition for which the patient seeks the surgeon's aid, but embraces as well the operative diagnosis and the recognition of postoperative conditions.

The essentials of surgical diagnosis do not materially differ from those of medical diagnosis in that the history of the patient needs to be most carefully taken and studied and the patient thoroughly examined. This is, as I view it, the keynote to diagnosis. I will admit that in certain classes of patients, it is not possible to obtain an accurate history. We must then depend upon our examination, our intuition and experience.

The interpretation of the history is the second most important factor. This must be done in a logical manner, and all the points brought out must be carefully analyzed in a deductive way. Patience and concentration are essentials if the translation is to be correct.

It has always been my practice to impress this upon my surgical internes. Often careful consideration of the minor points is of as much, if not more, importance than the more salient ones.

In eliciting a reliable history, first allow the patient to state his case, then follow with a series of careful questions,

* This lecture was delivered on Dec. 14, 1923.

none of which should be too leading. The manner in which the patient is approached tells for much. One must not be too serious, but must show a desire to shed all the light possible on the case so as to restore normal health. An antagonistic attitude on the part of the doctor cannot but thwart his efforts to obtain the truth.

In operative cases the history should be gone over with the patient some days after operation, during convalescence, when additions and perhaps corrections can be made, thus assuring a better and more reliable record. The patient's attitude of mind before the operation may be such as to render it impossible for him to recall all the points in his illness. I insist upon my internes reviewing their histories in this way.

After having made up one's mind as to the probable diagnosis, the next essential is the physical examination. In the abdominal case, this should consist first of inspection with the patient lying straight and flat upon his back. When inspecting the abdomen the contour of the two sides should be noted—whether there are abnormal prominences or depressions present on the one side that are not seen on the other. The patient should be asked to breathe moderately deep, also very deep with the mouth open; and observation should be made during both acts.

In suspected acute appendicitis, for example, in addition to the characteristic abdominal pain the diagnosis can often be further suggested by asking the patient to breathe moderately deep, or by having him take a very deep breath first slowly, then quickly; he will most likely tell you this causes pain in the lower right abdominal quadrant. Ask the patient to place his fingers where it hurts and he will place them over the appendiceal region. This localized point of pain can be further demonstrated by having the patient cough. This is equally true in acute conditions of the *upper* abdomen, but not so clearly demonstrable on account of the proximity of the diaphragm to the inflamed area. In the very acute abdomen, the abdominal walls are more or less or entirely immobile at the site of most intense inflammation and in its immediate neighborhood.

After having carefully inspected the abdomen, careful palpation, percussion and auscultation are next in order.

Palpation constitutes one of the diagnostician's most important assets. To palpate and thus elicit the utmost that can be learned is an art. Palpation is made by contact with the finger tips as well as by the flat hand. Lightness of touch is essential. It can be acquired, yet I question whether it is not inborn; however, we should aim to acquire it. Next to the history, palpation, percussion and auscultation are, to my mind, the most important methods of making a surgical diagnosis. I make this statement fully conscious of the value of the *x*-ray. But in acute, and especially in ultra-acute, abdominal conditions we are not able to make use of the *x*-ray. Palpation then serves to detect masses, swellings, irregularities, sudden movements, rigidity and flaccidity of the abdominal walls, tenderness, pulsations, the movement of gas, fluctuation, resiliency and tenderness, and in some instances hardening of the intestinal wall as the peristaltic wave passes along it, as in a hyperistaltic coil of bowel. Superficial and deep palpation with the fingers, as well as with the flat hand during natural and also during moderately deep or deep breathing, will often detect swellings and enlargements that otherwise might be missed, such as the movable kidney or the enlarged gall-bladder. This is made all the more possible if the examiner has a good working knowledge of anatomy, that is, familiarity with the topography of the various organs. I have many times stated that exquisite tenderness is a more reliable sign of the presence of pus than is a leucocyte count, and up to the present time, I have had no reason to change my mind. It has been well said that the best road to surgery is the road to and from the dissecting room. To this I would add the roads to the physiological laboratory and the post-mortem chamber. There is no greater essential to surgical or medical diagnosis than a knowledge of physiology carried to the bedside. To get the most out of the study of living pathology, one must have laid the foundation in the dead-house. It is usually too late to acquire this knowledge after one has become a doctor; this would be like making a version and delivering the placenta before the child. Here I may again incidentally remark

for the benefit of those directing the education of medical students, that the fundamentals of a sound medical education are in danger of being omitted, and in their place the accomplishments of the finished doctor are being taught; this is like eating the dessert first and the dinner afterwards. In my experience in teaching internes, I find they are rarely familiar with the dissecting forceps. Ask for dissecting forceps and you often will be handed tissue forceps. Partly in keeping with what I have said, they have been taught microscopic to the exclusion of macroscopic anatomy, and so down the roster. With an understanding of the anatomical relationship between the respective organs, palpation will be more enlightening. This relationship, however, can to a great extent be clouded by pathologic inroads, and then the knowledge gained by long service in the dead-house and in the operating theater, where living autopsies are being made daily, will chart the horizon.

Percussion is as essential to the surgeon as to the internist. The benefit of this aid to diagnosis is frequently obtained by shifting the patient from one position to another. It will not be necessary for me to dilate upon this further than to say that the sense of hearing is an important supplement, either used alone or with the aid of the stethoscope. This instrument, for those accustomed to its use, is perhaps the more reliable, and where there is abdominal tenderness it has the advantage over the direct application of the ear, in that there is less chance of increasing the patient's discomfort; in postoperative cases also, on account of the presence of the abdominal bandage, the stethoscope can be used with less annoyance to the patient.

Auscultation in abdominal diagnosis, and especially in the acute abdomen, is extremely useful if one is conversant with the character and site of the normal intestinal sounds and of the alterations in tone, intensity, etc. I hold it an indispensable aid in forming a correct conclusion of what is going on within. In the diagnosis of aneurysm, hearing the bruit clinches the diagnosis, while if there is not a true aneurysm, but only dilatation, not to auscult might possibly be to lose out, if the more modern means of making this diagnosis by *x*-ray is not applicable. The *x*-ray is a very pliable method,

yet it should not be used to the exclusion of auscultation, palpation and percussion.

In abdominal diagnosis, auscultation serves its most useful purpose to the surgeon in the diagnosis of the different varieties of peritonitis, as, with other signs and symptoms, it may assist him to decide in favor of immediate or delayed operation, as the case may be. In the presence of a peritonitis it is my practice first to auscult the abdomen and note the different types of peristalsis. In chronic intestinal and in early acute obstruction there is hyperperistalsis, while hypoperistalsis is heard in the presence of paresis of the muscularis of the intestines, due to infection. Tinkling is heard when the bowel proximal to the site of the obstruction is greatly distended, as well as where there is extensive paralytic distention—ominous physical signs which cannot be too much stressed.

Auscultation in peritonitis with the localization of the intraperitoneal lesion is of the utmost importance as an aid in determining the question of the time for operation. In the presence of a limited, diffusing and diffused peritonitis it will determine the absence of, or a diminished peristalsis over the involved area in comparison to that heard beyond. Immediate operation is indicated in the presence of a circumscribed peritonitis, with obscured or hypoperistalsis heard over the involved area, and with normal or perhaps slightly exaggerated peristalsis, distal to as well as immediately around the site of inflammation, and with a point of definite tenderness corresponding to the site of the lesion. These auscultatory findings in the case of a diffusing or a diffused peritonitis with the localization of the primary lesion also indicate operation. On the other hand, if, in the presence of a diffused peritonitis there is absence of peristalsis, a silent belly, in other words, with the pulsation of the aorta abnormally distinct, and inability to localize the original focus, operation should be deferred if the best results are to be obtained. The presence of tinkling, significant of paresis and a distended bowel, is often a serious sign and should tell the surgeon to stay his hand and await the result of anatomic and physiologic rest, i.e., lavage, Murphy drip, intermittent or continuous hypodermoclysis, etc.

It is in respect of the physical examination that the old school excelled; and why? Because sight, touch and hearing were for a long time the only available armamentarium of the diagnostician, and the man who had trained these senses to the utmost delicacy and refinement was the man of whom it was said: "He has the power of diagnosis in his finger tips." Men are still born with these same senses and with the potential development of "sight in their finger tips," but they do not sufficiently nourish, train and develop these gifts. For I maintain even today that inspection, palpation, percussion and auscultation still occupy a high place in diagnostic procedures. It is particularly in abdominal disease that the physical examination should be a complete one. The abdomen is a sort of loud speaker for many extraabdominal disorders. It is a well-known fact that pleurisy, pneumonia, pulmonary tuberculosis, endocarditis, cerebrospinal syphilis and a number of other diseases may be ushered in with abdominal symptoms. Differentiation of such conditions from genuine abdominal disorders can almost always be made by a thorough examination, particularly in case of the last-named, by examination of the reflexes, the presence of tremors and the like.

A proper interpretation of the history and the results of a complete and careful physical examination is, as a rule, a sufficient guide-post to the correct path of diagnosis. But it would be about as unwise today to rely upon these alone, without the aid of the laboratory, as it would be to send a messenger boy when a telephone conversation is available, or, to be ultra modern, to go by automobile when an aeroplane is at one's disposal. For the laboratory has come to play the great part of confirmation where formerly hesitancy existed. Not only that, but it is in the correlation of clinical with laboratory findings that the laboratory has added to the sum and substance of our knowledge.

And yet, we must use considerable judgment in the interpretation of the laboratory findings. Unfortunately, many of the tests designed to assist in diagnosis are not of as much value as their enthusiastic supporters claim for them; and as I have often remarked, are frequently more impressive to the laity than of value to the surgeon. There is no doubt that the

fractional test meal is more reliable than the simple test meal which formerly enjoyed more extended use, but the acidity of the gastric contents deviates so often from the expected that it has not much value as a guide. We cannot specify what sort of curve for gastric acidity is entirely normal. Our experience with fractional test meals, as checked by operative findings in many hundreds of cases, leads to the general conclusion that both in the absence and the presence of anatomical change in the upper abdomen the acidity may be low, moderate or high. For example, the free and total acidity were within the "normal limits" in 25 per cent of our cases of gastric and duodenal ulcers, while in 13 per cent there was subacidity. In disease of the gall-bladder our figures show almost equal percentages for hyperacidity, subacidity and "normal" acidity. This is to be expected considering the numerous factors of error creeping into determinations such as these. The finding of occult blood, either in the stomach contents or in the feces, is another very uncertain aid for the same reason of the factor of error.

Of more value to us is the leucocyte count; in fact, a complete blood count is an essential routine procedure for all surgical cases. Diagnostically, it reveals the presence or absence of anemia, a question of considerable importance in certain cases. The balance in favor of operation is often swung by the finding of anemia and high leucocytosis in a number of cases of doubtful ruptured ectopic pregnancies. I refer particularly to those in which the bleeding has not been rapid, but the patient's symptoms have been spread out over a period of a week or ten days. In septic conditions the leucocyte count is very often a reliable index, but attention must be called to the fact that a leucocytosis of high degree is not apt to be manifested when a septic process is well walled off, and the body as a whole does not take part in the reaction. I have seen numerous abscesses originating from appendicitis, in which the leucocytic count has been normal, or practically so. These abscesses were walled off quite securely, and my diagnosis depended upon what I have before mentioned as exquisite tenderness and other signs, such as hindrance to respiration, and so on. But when all other signs fail, leucocytosis does occasionally warn

us that trouble is present. I have in mind a recent case of a man, aged thirty, who came to operation for acute appendicitis. His appendix was enlarged, stiffened and congested. Its peritoneal surface was dulled and there was considerable exudate in the mesoappendix. No drainage was instituted. Recovery was uneventful for a period of five or six days, when a slight degree of fever gave warning that all was not well. The leucocyte count reached 20,000. There were no subjective symptoms of any kind present, nor did the most careful physical examinations, repeated a number of times a day, demonstrate any objective symptoms whatever. The patient declared that he felt perfectly well, was hungry, and his other functions were working satisfactorily. Three weeks later, with persistence of the high temperature and the high leucocytosis, he suddenly developed severe pain in the right hypochondrium with palpatory findings of tenderness and rigidity. The second operation revealed an acute suppurative cholecystitis. Unfortunately, ascending infection of the liver was also present and the patient quickly became jaundiced, but happily, he has made a good recovery. The danger signals he flew were only two: slight fever and persistently high leucocytosis. Diagnostically, therefore, a blood count is of great value, but it must be correlated with other factors in the case.

From the standpoint of judgment, the laboratory is an invaluable, I might say indispensable, aid to the surgeon. Its usefulness lies both in specific cases and in generalizations. In the case of each individual patient, the question of the factor of safety for operation should be considered. I need not call your attention to the fact that anesthesia alone is a procedure sufficient to place certain individuals in grave danger. From the standpoint of the surgeon the excretory system very often renders an otherwise rational and successful procedure disastrous. It is our practice routinely to examine the functional ability of the kidneys. This we do by means of the phenolsulphonephthalein elimination estimation and the quantitative determination of the blood urea. By these tests, in conjunction with an ordinary urinalysis, we are able to forestall kidney complications, and often when they indicate poor functional ability, I

allow the patient to remain in bed under treatment for some length of time before undertaking an operation.

In diabetics the laboratory is the keystone of the arch of treatment. From the knowledge derived from the blood sugar and urinary sugar determinations and an occasional blood bicarbonate estimation, and with proper diet, we are enabled to operate upon these patients with as good hope of success as on non-diabetics. The advent of insulin, as Allen says, "while it has not simplified our treatment of diabetes, has most certainly placed a very powerful tool for its management in our hands."

From the general standpoint, therefore, the laboratory is indispensable, providing as it does accurate and definite knowledge in the fundamental branches upon which diagnosis rests. I refer to anatomy, which I have already emphasized, pathology, bacteriology and physiology. I, of course, use the word "laboratory" in this connection in its broad sense. Take, for example, the case already referred to: when our patient developed a septic temperature and jaundice, the knowledge derived from the anatomical relations, the bacteriologic possibilities and the pathological study enabled us to make our diagnosis with almost certainty. To put it tersely, he is the best diagnostician who can combine his twos and twos and calculate the total.

ESSENTIALS OF OPERATIVE DIAGNOSIS

In operative abdominal diagnosis the essentials are a knowledge of the anatomy of the viscera, structural and topographic, of morbid anatomy, living pathologic anatomy and surgical physiology. We all appreciate how difficult or comparatively easy an operation may be, depending upon this knowledge. As an illustration, I can cite the removal of an appendix not the site of extensive pathology; how often have I seen surgeons consume much time and perspire freely in doing such an operation. On the other hand, recognition of the cecum, and perhaps being able to lift it into the wound, and identification of the ileocolic and ileocecal folds at once enables the operator to follow the latter to the mesoappendix and the appendix. The appendix may be buried in either the ileocecal or subcecal fossa, and when in the latter, if the

margins of the orifice of the fossa are adherent to the base of the appendix at its junction with the cecum, it may be overlooked entirely. Confusion also may arise when the appendix is retrocecal or retrocolic and covered by a pathological sheet of peritoneum.

Anatomic landmarks, particularly in the presence of a clear horizon, should make recognition very easy, and in the presence of a foggy atmosphere, give confidence and help which facilitate operative procedure and, therefore, make it safer. A knowledge of dead-house pathology is the foundation for the subsequent structure of living pathology. I often question whether the modern tendency is not to acquire anatomic knowledge exclusively from the living instead of from the dead subject; this is not in any way of advantage to the patient.

Indeed, it is not always easy with the abdomen open to verify the pre-operative diagnosis, assuming this to be correct; nor is it always easy to make the correct pathologic diagnosis if it has not previously been made. The essentials in both are the proper exposure and recognition of the organs or tissues involved with the minimum amount of traumatism.

In the upper right abdomen the most common lesion is first, disease of the gall-bladder, and second, disease of a high-lying appendix. The essentials in the operative diagnosis of either are a good exposure and a knowledge of the pathology of both. Pericholecystic adhesions, as a rule, mean a diseased gall-bladder, but not always. Inflammation of an appendix in a high position causes a peritonitis which does not always resolve without leaving reminders of its presence, such as sheets of pathologic peritoneum, etc. These in time become attenuated and thinned out, and form adhesions which usually can be identified with the particular organ responsible for their formation, only by exposure and careful examination. In the operative diagnosis one condition is frequently confused with the other, as I have many times proved at the living autopsy. A not uncommon *x*-ray diagnosis is a pylorus in a high position and adhesions. This of itself means but little, but it is frequently interpreted as suggesting disease of the duodenum, especially if deformity of the duodenum is also included in the *x*-ray diagnosis. In my

experience, these findings indicate pathology either of the gall-bladder or of the appendix. I find that, with very few exceptions, a nonperforated duodenal or prepyloric gastric ulcer does not cause periduodenal or perigastric adhesions.

In the operative diagnosis also of the liver, the hepatic duct, the common bile duct and the pancreas, and diseases of the gall-bladder, good exposure is essential, in order carefully to inspect and palpate. In the case of the biliary ducts, palpation and inspection alone will not always suffice, as, for example, in the presence of stricture or a stone in the common duct in relation with the pancreas or the walls of the duodenum; here incision into the duct and instrumentation must be added to inspection and palpation. The diagnosis of certain forms of jaundice, as well as pancreatic lymphangitis, or chronic pancreatitis and retroperitoneal pathology can be made with certainty only by this method.

The pre-operative diagnosis of ulcer is confirmed or disproved by operative inspection and palpation, and in a few cases, with incision through the anterior wall of the stomach and wide retraction. The latter is occasionally necessary for diagnosing a possible marginal gastrojejunal ulcer. With the interior of the stomach exposed through an incision in the anterior wall the stoma of a gastroenterostomy can be readily brought into view and carefully inspected and palpated. The *x*-ray diagnosis of duodenal diverticulum, of duodeno-jejunal and high jejunal carcinoma, as well as a lesion of the intestinal wall elsewhere, can be made with certainty only by opening the abdomen.

The laboratory in operative diagnosis is a distinct aid. First and foremost are the results obtained by the frozen-section examination of tumors removed at operation. I have said that a knowledge of morbid anatomy is essential to operative diagnosis. With this knowledge it is, of course, possible for the surgeon to distinguish at least grossly between malignant and benign tumors. He should also, in the large majority of cases, be able to distinguish grossly between epithelial malignant tumors and those of the connective tissue type. But it is in the border-line cases that the frozen section has its almost indispensable use. No one, be he sur-

geon or pathologist, even with the maximum of experience, can always decide between a malignant and a benign tumor in the gross specimen, and it is just in these cases that the real hope of complete recovery can be entertained when the removal of a malignant tumor is complete. We are happy to say that our frozen section diagnoses have been confirmed by later study in between 98 and 99 per cent of cases.

THE ESSENTIALS OF THE DIAGNOSIS OF COMPLICATONJS ARISING AFTER OPERATION

The essentials of the diagnosis of the complications arising after operation are of equal importance with those before and during operation. Prominent among these are: The frequency and type of vomiting and the character of the vomitus; the condition of the abdomen, whether soft, rigid or distended; the presence and the character of the peristalsis whether hyper-, hypo-, or tinkling, or its absence; the amount of urine voided, its character, particularly its specific gravity, the presence of sugar, acetone or diacetic acid; the appearance of the face and the activity of the mind; the character and rapidity of the respirations; the pulse and temperature.

What has been said of the value of inspection, palpation and auscultation in the pre-operative diagnosis holds equally good in the diagnosis of postoperative conditions. Another essential in the postoperative diagnosis is lavage. Where there is suspicion of a bowel obstruction, and pain is already present and not of the intermittent type, I regard lavage of great value, indicating the presence of duodenal or upper intestinal regurgitation. We learn to anticipate certain surgical conditions by careful scrutiny alone. The color of the patient often means much to the surgeon, for example, the blanched lips in severe intra-abdominal hemorrhage and the breathing, etc. Digital examination of the rectum is often essential. For example, how often will it reveal a fecal impaction when least expected by the internes and the nurses; again, how often will passing a catheter and withdrawing a large amount of urine dispel mystery in the shape of an hypogastric tumor; or result in the immediate disappearance of frequent urination.

The more common complications after operation include shock, hemorrhage, pneumonia, cardiac weakness with tachycardia, peritonitis, mechanical intestinal obstruction, paralytic ileus, secondary collection; and, during convalescence, pulmonary embolism and acute cardiac dilatation.

While it is true that shock after surgical operations is now seldom seen, it occasionally occurs. The essential points in the recognition of shock are the rapid and compressible pulse, the extreme weakness and apathetic state of the patient, the relaxed skin, the low temperature and the low blood pressure. It may occur before the operation is completed. Occasionally shock is delayed for a short time after operation, but it does not differ symptomatically from that occurring during or immediately after operation.

Internal hemorrhage is recognized by the blanched appearance of the patient, restlessness, the rapid, small, compressible pulse, air-hunger and thirst. It is not always perfectly clear whether the patient is only shocked or is bleeding as well. The diagnosis cannot always be made until after an intravenous salt solution which, with few exceptions, immediately brings up and maintains the pulse in shock, but which it does not do when the patient is bleeding; a complete blood count is essential. Here lies the importance of routine blood counts, for we cannot always tell which patient will and which will not bleed. Cardiac weakness with tachycardia clouds the picture. Unfortunately, the administration of the different cardiac stimulants does not always give the hoped-for relief. This is particularly true of digitalis; nor should we expect to get an immediate effect from this drug. I derive better results with morphin and a small amount of salt solution in the vein, than with the usually recognized diffusible stimulants, including caffeine, pituitrin, camphorated oil, etc. When I was a hospital interne, whiskey and ether were given; and I still occasionally adhere to a habit of my youthful days.

Peritonitis occasionally follows, or is aggravated by, operation. The essential points in the diagnosis are nausea, soon followed by vomiting; pain and tenderness with greatly diminished or entire absence of peristalsis over the area of most intense inflammation; rigidity of the abdominal wall at the same site, soon followed by distention, elevation of

temperature, increased pulse, increased abdominal tension and inability to pass flatus.

When peritonitis is well advanced, the picture consists of paralytic ileus with regurgitation of the contents of the upper small intestine, uniform distention of the abdomen, inability to pass gas, tinkling upon auscultation and loud pulsation of the aorta. Accompanying these signs are: a bright eye and an active brain and, not infrequently, superficial cyanosis. The patient, however, does not realize the gravity of his condition.

Intestinal obstruction, of either the slow or the rapidly forming type (more often the former) causes, first, intermittent cramp-like pains, immediately followed by nausea and later by vomiting, hyperperistalsis, and later still, absence of peristalsis and inability to pass gas. When the abdomen has been opened in the presence of infection, mechanical obstruction may occur without giving the classical symptoms. The diagnosis of obstruction can be made on the absence of fever, with at first a practically normal pulse rate, and later, a rapid, weak pulse, marked distention without marked tenderness, the absence of peristalsis, accentuated pulsation of the aorta and either vomiting or regurgitation of dark material with a fecal odor. Lavage will wash out large quantities of the same material, and no matter how often repeated, the same result will be obtained. If lavage is not followed by reduction of the abdominal distention and cessation of the vomiting, the abdomen should immediately be opened.

A perplexing condition is secondary abscess, often associated with complete obstruction, for which operation is the only solution. We have learned by experience that localized tenderness, distention and fever, with peristalsis present, are the cardinal signs of secondary abscess, and that these abscesses show a preference for certain situations, namely, at the site of the original operation, or in its immediate vicinity; in the pelvis; behind the cecum and colon in the sub-hepatic space and between the diaphragm and liver. Occasionally, a secondary abscess is centrally located. In the diagnosis of sub-diaphragmatic collections the *x*-ray is of great value in determining immobility of the diaphragm in the presence of a collection immediately beneath it.

Infection of the incision can usually be recognized by the absence of marked general reaction, the presence of tenderness, fullness, etc., the signs of inflammation in the wound itself, plus great abdominal distention at times.

The diagnosis of postoperative pneumonia is also one which usually presents little difficulty. The reaction of the patient includes rapid and labored respiration, together with the physical signs of consolidation, which may, however, delay their appearance for several days. The question of differential diagnosis between what might be called pure bronchopneumonia and pneumonia in localized areas following the transport of infected emboli, is often difficult, and not infrequently the distinction must be postponed until later in the disease.

The laboratory is of value in the diagnosis of all these postoperative conditions. Our first thought is directed toward the blood count. In cases of hemorrhage the comparison between postoperative and preoperative count is worth all the routine work necessary in order to have this index of comparison ready when occasion demands. The leucocyte count also, quite frequently, with the aforesaid limitations, tells us of the presence or absence of infectious processes.

As to bacteriology, the usefulness of cultures and smears from our glass tubes can be extended to wounds; oftentimes we have had occasion to prepare autogenous vaccines, and we believe that, in certain cases, the results have justified their use. The results of smears made during operation indicate what to expect after operation, and enable us to decide whether or not to drain; also we may give the friends of the patient a more definite prognosis.

Although acidosis does not play the part in surgery that was ascribed to it some years ago, it is nevertheless, in a certain number of instances, an added burden to an already overtaxed organism endeavoring to recover. By means of determinations of the bicarbonate content of the blood, we not only are enabled to determine the presence and degree of acidosis, but can actually calculate the necessary dosage of alkali to restore the acid-base function of the body to a normal level. In diabetes, the laboratory is, of course, a first aid in the postoperative management of patients.

I need hardly call your attention to the indispensability of accurate pathologic descriptions and reports of tissue removed at operation. In many cases they establish the diagnosis; they determine the future mode of treatment, especially in tumors, but no less so in certain other types of disease. I might mention as an example, tuberculous salpingitis. This condition is by no means uncommon. Naturally, it requires a different line of treatment than do the ordinary acute or chronic suppurative conditions. A careful pathologic examination with the help of the microscope is often the only way of positively determining the presence of this specific infection in these and other organs.

Such a simple thing as an ordinary urinary analysis is oftentimes the pivot around which postoperative therapy revolves. In the case of pyonephrosis and cystitis, when the offending kidney is removed, we expect the cystitis to disappear. The urinary analysis determines the diminution in the quantity of pus as the pathologic process is overcome.

Finally, from the standpoint of research our operative procedures have oftentimes given the stimulus to the laboratory to investigate functions of various organs of the body when they are placed in other relationships. Fortunately, advantage has been taken of many such opportunities and the benefit has been mutual. Not only has the surgeon in this way been a stimulus to the laboratory worker, but the laboratory worker has, in his turn, unearthed facts in the physiology of the various organs which have reacted to the benefit of the surgeon, and thus directly to the patient. By this team work, therefore, much valuable knowledge has been gained. Let us hope that this cooperation will continue. The benefits derived from it will most surely make themselves felt in the increasingly better interpretation of what our trained senses tell us; in better and more accurate aids to our senses, and better and more comprehensive help to our patients.

OFFICE GYNECOLOGY*

ROBERT L. DICKINSON, M.D.

THE time is nearly due for the two sisters, Office and Operative Gynecology, to take thought on the matter of the inevitable exchange of their rôles. The small one, the showy junior, now has the suites and great apartments in stately buildings, the money and the headlines; yet it is the senior, dwelling in two rooms or a basement, and providing the infinitely wide-spread, quiet service and the decisions on life and death, to whom the future belongs. For when Organized Preventive Gynecology comes into its own, and large tumors become curiosities, cancer is all but prevented, and obstetric injuries have been forestalled or at once repaired, and girls and women are regularly examined as a matter of general precautionary common sense, then the operations of our period, in their degree and multitude, will be classed as rank neglect.

Thus the major of today is the minor of tomorrow.

Gynecology as a department of medicine is peculiar to itself in its simplicities and size. The disorders on its lists are few in number, but infinitely repeated and therefore conspicuous by their totals, while their length of range extends from many "functional derangements" to some of the gravest risks in surgery. Though its operations comprise more than one-fifth of major surgery, the hospital proportion bulks less than one-tenth of the whole. The nine-tenths it is our brief business to touch upon, though in attempting this summary a serious difficulty is inherent, owing to the differing points of view of the two parts of the audience. One section is made up of general practitioners interested in diagnosis and those simpler treatments within their scope, and the other of surgeons and specialists who wonder whether there will be brought forward any little tricks one has overlooked or forgotten. In riding the two horses the speaker has to do no little straddling.

*This lecture was delivered on April 7, 1922.

The time is apt for looking forward. Abundance of new information and wide extension of a new mental attitude on the part of women are busily changing certain aspects of our work. The war gathered a mass of evidence which was sorted mainly in England and Germany, showing what a woman can carry, in hours and weights and monotony, and at her periods and in pregnancy. The war tested the corralling of the genitally infected: it demonstrated that much venereal disease radiated from a countable number of focal points, and it developed other new coital and venereal data. Moreover, it came to pass that a nation listened to sex hygiene, and listened without the awaited shock. To the female student, outnumbering the male as she does, have been given the beginnings of systematic instruction in sex matters. Birth control information has spread widely, notwithstanding the refusal of the medical profession to countenance any study of the subject.[1] The hygiene of the young woman has been under investigation in colleges, with such experiments as exercise and cold baths during menstruation. Insurance companies and corporations have collected medical data about women. And, most far-reaching and method-searching of all, hospital-staff reviews and outpatient follow-ups have begun a nation-wide scrutiny of the end results of operation; so that the day is to come when we shall everywhere install surgical accountancy, and officially list surgical insolvency.

In taking account of stock in our specialty we find on the shelves a number of devices in the way of delicate, expert manipulations and a variety of resources in non-operative treatment that are in danger of becoming a lost art—lost in two or three decades, with the office the mere antechamber of the ether room—and a former swift and tenderhearted technique blunted by the present habit of dealing with a patient temporarily dead to suffering and shame. We shall restudy, therefore, the details bearing on privacy, on unsensual methods in nudity for thoroughness, and on the minimizing of discomfort and pain in "minor" gynecology.

[1] Up to 1923, when the Committee on Maternal Health took hold.

EXPEDITION IN OFFICE PRACTICE, OFFICE PLANS AND DETAILS

A doctor is handicapped beyond all other men in professional and business careers by being only a one-man power. He is able to delegate little. Organization of work by unloading on partners or assistants finds less play than in other livelihoods calling for equal brain action. The more need, therefore, of system and intelligent planning in whatever matters these may expedite and simplify. A man in whose

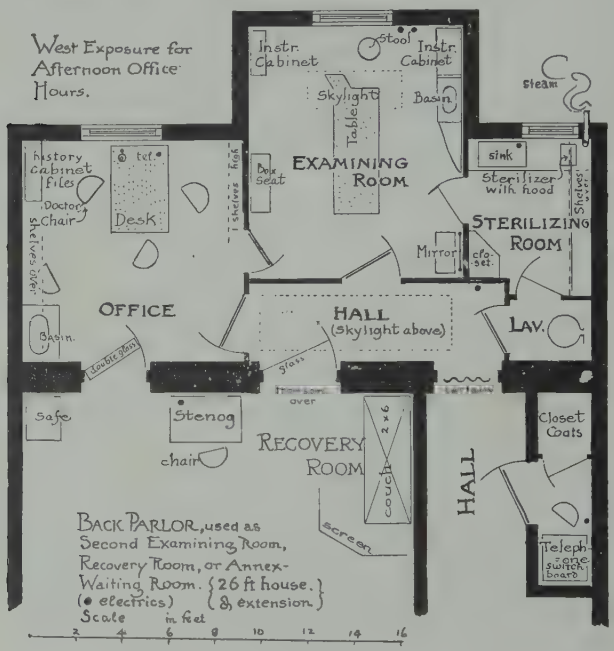


FIG. 7.

operating room and hospital wards complicated machinery works with smoothness and celerity often fails to engineer the details of his office work with the same executive skill. It is the sense of hurry, the consciousness of impatience on the part of waiting people and particularly the chafing at needless delay, that wears on the man in the office. Even for the beginner facilities for quick work will pay. Though he have only a few patients, they have a fashion of bunching

at the start or at the finish of his office hour, and every one of them begrudges the time lost in the waiting room.

In no way can the doctor save more time than by wise arrangements of his quarters. The different offices should be accessible from the hall without passage through each other (and without necessarily passing through the waiting room), so that entrance or exit is possible for each independently. (Figs. 7 and 8.)

The desk office should be separate from the examining room. For the busy man two small waiting rooms are better

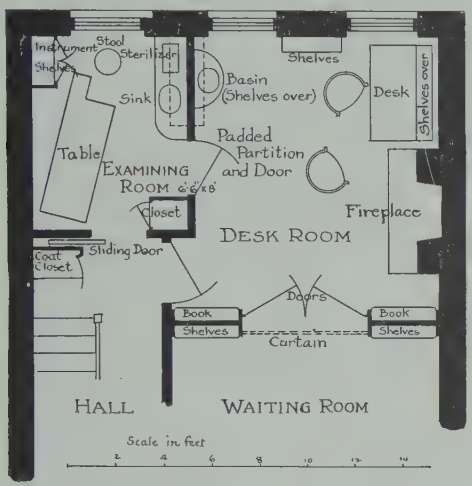


FIG. 8. Office in ordinary 18-ft. city house where front parlor is waiting room, showing how a nearly sound-proof examining room is divided off, with its own exit. Commode is under examining table for emptying bladder.

than one large one; two small examining rooms better than one large one. (Fig. 8.) The offices should be located in the natural order of progress of the patient, viz., hall, waiting room, desk office, dressing room with toilet room, examining room, recovery room or dressing room, hall. (Figs. 7 and 8.) Ten feet square is a minimal, but sufficient floor space to accommodate a single office (as distinguished from waiting room or dressing room). The water-closet and the telephone closet should be accessible from both waiting room and offices, preferably by way of the hall. Walls and doors should be measurably soundproof. Doors should be hung so

that, when partly open, they screen the occupants of the room, and not primarily for appearance or convenience in entering.

In two different offices I have seen walls efficiently deadened at small expense by ordinary, cheap, wadded quilts placed between two thicknesses of wood. The same material tacked on a door and covered with painted canvas is very satisfactory. As with all doors, weather strips are necessary. Double sliding doors are difficult to deal with.

The doors between waiting room and office should be padded on the inside or hung on one or both sides with relatively heavy curtains, and weather strips should stop the cracks. Doors may need to be rehung so that a patient on the table or with bared chest will not be in sight when a knock calls the doctor to the door. A screen or curtain enables a patient to dress under cover. It conceals the suggestions of trouble to come—the examining table, the instrument case, and also the disorder left after examination or treatment. Even in an examining room a cubicle of a dressing room is desirable. In our Brooklyn Hospital dispensary two doors, so hinged as to box in a corner, offer to every woman this privacy in each examining room.

The ordinary house in city or suburb or country is susceptible of alteration at moderate cost. In these days of electricity, interior rooms have become available by the use of a method of illumination that does not vitiate the air, aided in appearance and cheerfulness by light wall coloring and bright lighting, by the headlight that is always better than window light, and by the use of the electric fan.

Desk Office. The few requisites of the history room, to wit, desk, chairs, book shelves and provision for histories and accounts, make this the least important of the offices. Yet to it is often given space more needed for the equipment required for examinations and treatment. It is rare that there are in the desk office more people than the doctor, the patient and one or two others; therefore four seats suffice. Much room may be saved by placing the book shelves so that the lowest shelf is five feet from the floor. This will leave all the wall space, except where doors and windows come, available for chairs, desk and passage room, and at the same time pro-

vide generously for books. Three tiers of books may thus be within reach. It should not be forgotten that a shelf nine inches deep will hold big books.

Examining Room. The best lighted, best ventilated room should be chosen, preferably one facing north. Direct sunlight is desirable neither in summer nor in winter. Therefore the man with the morning office hour, if he must choose

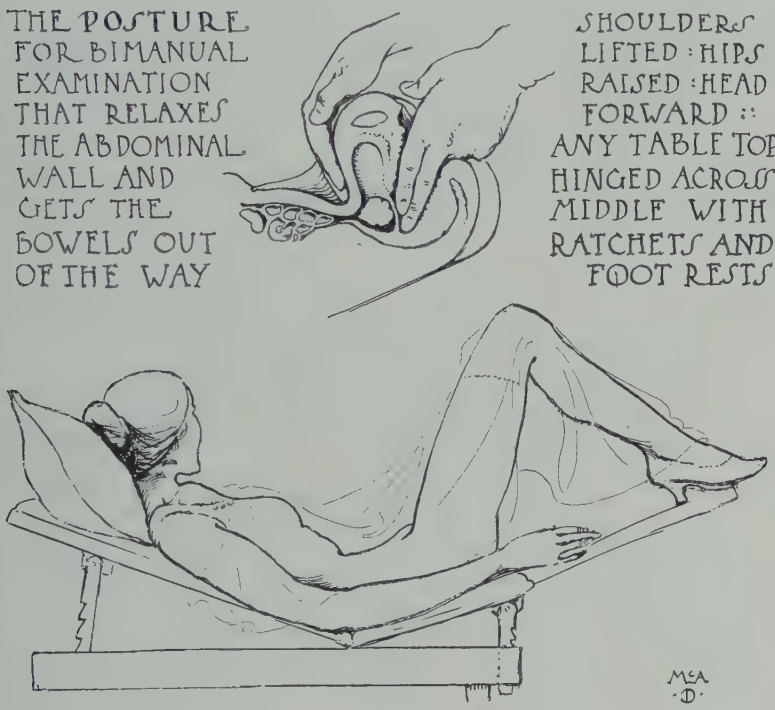


Fig. 9.

between east and west, should face west, and the man with the long afternoon hour face east. A light finish on wall and ceiling reflects and diffuses light, and is economical in night illumination. A washable wall, painted or tiled, is best. The light-tinted and enamelled woodwork should be warm in tint—cream white, for example, not bleak blue-white or a cold green. Glass must cover the tops of stands and tables. Milk tint glass is good.

As to the table, individual preferences will always rule. It must be steady, yet is best on large rollers, to be moved about readily. It must be comfortable to lie on, yet easily cleaned. For simple forms I prefer enameled wood for its steadiness and lightness (Fig. 9). Patients are somewhat fearful of iron machinery. The best of these is the Boldt table (Fig. 10). Its top adjusts to a variety of inclinations, desirable because a large proportion of pelvic examinations include the bladder and rectum.

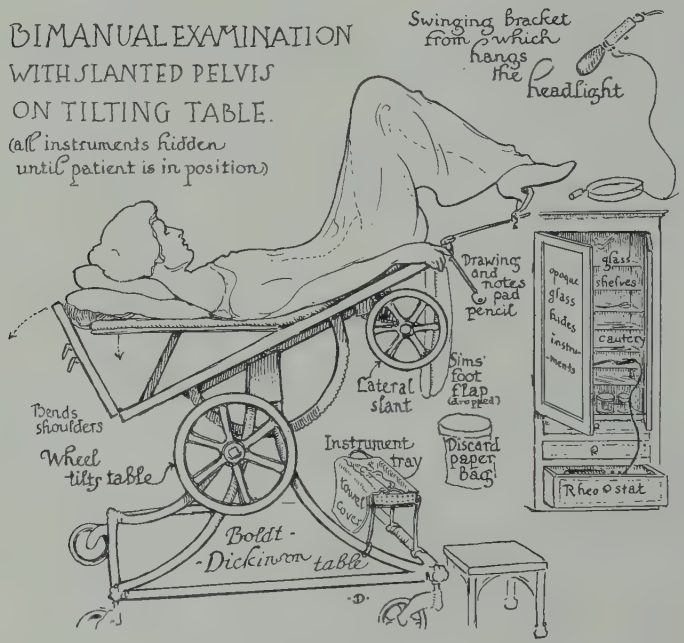


Fig. 10.

Artificial Light vs. Daylight. It holds as true for the gynecologist as for the man who deals with the larynx or the ear drum, that artificial light is better than daylight. And for these reasons: Looking into the vagina, we look into a dark cavity whose surroundings are necessarily pure white. Now the more brilliant the light that falls upon the coverings of sheet and towel, the more the pupil contracts and the less it can make of the non-actinic red in the cavity. Let but direct sunlight fall upon the sheet and the cervix disappears.

Thus this paradox holds good—the more light, the less one sees. Moreover, when light from a window is used, the patient must adapt herself to the direction of the light. With the head-lamp the light follows about and adapts itself to the patient's position, and may sweep around this cavity which is narrowest at its entrance. Through a hymen so narrow that the finger-tip cannot pass we may painlessly treat a raw cervix, take off a polyp or dilate a cervix, because we have perfect illumination, tiny specula and a skill born of training in difficult bladder work. The wire to the head-band should fall to it from above. To place a light for the headmirror over against the operator, a good method employs a light arm swinging from the wall, so jointed that it stays in any desired position. The Faries bracket used by dentists, to be had of the S. S. White Dental Company, is entirely satisfactory and is cheap. There are also brackets for a lamp or a Welsbach or Argand burner, but these are less convenient.

Duplicate Outfits. To save time there should be two or more sets of those instruments which a surgeon or specialist needs constantly. Then one set may be in its tray in the sterilizer while another stands in its own tray or rack, ready for use, properly covered with a sterile towel or paper napkin (Fig. 10).

Sterilizing-laboratory-supply Room. A space not much wider than a corridor—enough for a shelf, a chair before it and for passage behind the chair—will serve for the unkempt room of the plan. Here, in shelves above, supplies are kept, with doors, not hinged on the side, but pivoted or hinged to slide sidewise or turn upward, protected from dust.

The sterilizer with its steam is best kept out of the offices, even if it has to go into a box of a closet. Wherever it is placed, it should have a hood and a pipe leading outdoors through wall or window to carry off steam and heat. The cost is slight, the comfort great.

Toilet-closet. A minimum size for a water-closet is 2½ by 3 feet. (Fig. 7.) One 3 by 4 feet gives ample space. If it has two doors, one opening from the hall and one from the office (Fig. 7), they can be locked at one motion by the person within by means of a long hook reaching from the inside of one door to an eye on the inside of the other. Neither

door of the toilet room should be in full view of hall or waiting room, nor should it open only into the examining room or office; otherwise persons who are waiting will not have access to it. With no toilet, a commode or drawer in the table may be put up with.

One closet may wisely be adapted for a telephone booth. A coat closet may be so used; or a corner of the hall is easily cut off diagonally to close in the telephone. Many of the doctor's messages over the wire are of a nature that renders it desirable for him to be able to shut himself in. The desk set may be on an extension of the same line. Furthermore, this telephone in a hall closet makes it unnecessary for servants to enter the office to answer calls during the doctor's absence (Fig. 7).

Recovery Room. It is supposed to be unwise for a doctor to have a couch of any form within the closed doors of his office. At any rate, it is usually unnecessary. In the rear of the waiting room a screen drawn around a narrow couch or divan gives a degree of privacy and quiet; or the end of a hallway will do. A part or all of what constitutes the secretary's quarters during other parts of the day may be used in this way (Fig. 7).

Assistance and Nursing. By doing special work only on special days, many young men can afford assistance. It should be afforded before it can be afforded if one is to do gynecology. In some cases a discreet housemaid acts as office nurse. At the next step the attendant becomes stenographer, accountant, secretary and nurse in one. For office work a conscientious woman who has learned shorthand can easily be taught surgical cleanliness and the keeping of accounts, because the round of duties is a narrow one, the possibilities of error few and the supervision constant. The experience and training and high salary of the hospital graduate are not necessary as a preparation for ordinary office work unless the office nurse is called upon to assist at outside operations. There is all one nurse can do in a busy one-man office, as of a gynecologist, in the care of offices, instruments, supplies and dressings, in the preparation of the ever-ready operating bags, in keeping the accounts and typewriting letters and manuscript, to say nothing of attendance at the door and

the telephone outside of office hours. She may be trained to the routine of urinalysis, staining, hemoglobin color test, and the like.

Automatic Office Moralities. The mechanical safeguards that furnish protection to the patient act also as shields to the doctor, and should be a matter of considered thought with the gynecologist beyond most men. He equips his office with sound-proof walls and weather-stripped door-cracks in order to muffle the confidential communications of the shrill-voiced, and to enable him to question or instruct the deaf woman. He forestalls criticism of such measures by arranging to have no latch on any door that would hinder entry at any time. The attendant is encouraged to come in freely with message or errand after knocking and receiving sanction. In no inner room is there a couch. There may be in each door a window of two layers of rolled or "cathedral" glass to prevent the sight of any definite form, but to permit vision of the presence and general position and movement of those within the room. Furthermore, in vaginal examinations the doctor asks whether he is hurting and has the habit of seeing to it that there is associated with examination and treatment enough discomfort or pain to ensure the absence of the opposite sensation.

The chief provision, however, for automatic protection of all concerned, which is taken by the man who is obliged rather often in the course of his duty to be the recipient of information or the questioner concerning certain items of sex behavior, feelings, habits, responses or risks,—the chief safeguard is the inevitable presence of a third person. Husband, relative, friend or office nurse is in sight or hearing or just beyond. This rule may be waived for the history taking or final instruction with particular secret troubles, but it is inflexible during the examination of women by men. Herein, as in all the above provisions, one is not in the attitude of offering insult to his patient or himself by an arrangement which would appear to presuppose possibilities of evil in thought or word or deed,—one is simply installing a standard form of decent care.

Printed History Form Filled Out at Home. Any doctor who cares to be thorough can save considerable time by

having the patient work out her own history, if she is of fair intelligence. The form[1] I have published covers, on four pages, (8½ by 11 inches) a full statement of symptoms and of illnesses, operations and deliveries. It is sent to the patient when her appointment is made (or she takes it home). In actual practice it is found to require four or five minutes of the doctor's time to read and to redpencil. The essentials are underscored and are then or later copied by a secretary or oneself into the summary of the patient's history, placed at the end of this sheet or on the separate history card. To ask the equivalent questions and to write down the answers (using or not using such a history sheet to do it) takes at least twenty minutes. But even this is not the main question. There is no such thing as a patient who can give her history in order or without important omissions, or without a good deal of consideration. This means that a real history can be made rightly at home only, as it usually requires conference with other members of the family. Moreover, the names of doctors who have cared for the patient in previous illnesses are not to be had offhand. And lastly, the patient hereby acquires a concept of how careful our studies of her story must be.

EXAMINATION

Failure to diagnose arises more commonly from failure to run a routine than from any other cause. The omissions are chiefly insufficient removal of clothing and defective emptying of the bowel or bladder, and lack of employment of the best postures either in bimanual or speculum examination, together with forgetting that pelvic pains may be due to the articulations and to bowel and bladder. We have to remember that it is not enough to determine the presence of a disease or disability or growth which amply explains the complaints of the patient but that one or more complicating troubles may be present. Treatment of or operation for only one of these things when the others might have been discovered and removed at the same sessions is not a matter for which one can make excuse. This is particularly true among women with their intestinal disturbances and their

[1] Dickinson, Robert L. The new "efficiency" systems and their bearing on gynecological diagnosis. *Am. J. Obst.*, Dec., 1914, lxx, 865–884.

nervous systems disordered or exhausted. The orthopedist, the gastroenterologist, the neurologist and the urologist are important members of the firm, and a certain amount of knowledge sufficient to indicate when to refer patients to them is essential in office gynecology.

The patient's clothing is to be thoroughly loosened, and the corset, if any, is to be entirely removed. The bladder is to be emptied. If the bowel is found to be in any degree loaded, it is imperative that an enema be taken at once or that the patient return after a good clearing out. One not infrequently declines to make a complete diagnosis wherever the lower abdomen is other than freely accessible; one declines to give a complete opinion until a second examination has been made. This may be offered as part of a single examination fee. Some day we may decline to reach conclusions unless checked up by a second examination.

As the patient steps upon the footstool ready to sit upon the table, the sheet is raised by the nurse (or doctor) as a screen, so that the patient may lift her clothing before sitting down, and, after she sits, the sheet be thrown across her legs and trunk. Shy patients sometimes prefer to have their faces covered. The paper napkin is already over the pillow; the double paper napkin is now slipped under the buttocks, the feet being properly adjusted in the stirrups (or on the knee trough leg-holders).

The examination, of course, begins with teeth, tonsils, thyroid, breasts, nipples (heart, lungs, blood-pressure) and upper abdomen, before looking for protrusions and tendernesses, scars, striae or some form of hernia about the lower abdomen.

The bimanual to be reasonably effective calls for a relaxed abdominal wall and for a minimum amount of bowel in the pelvic and lower abdominal cavities. This is to be secured by bending the chest and head moderately and tipping the table with the pelvis elevated (Figs. 9 and 10). Every gynecological table or chair should permit of this arrangement. The figures show that it can be done with the kitchen-table type that the beginner can afford, or it can be done with the ratchets of the complicated table. The only table that does not permit convenient gynecological examinations is

the surgical table with the solid, full width lower section that drops down at right angles. Without a narrow shelf, to hold the lower legs in Sims' position, which projects sufficiently far from the left-hand corner of the table as the doctor faces it, the best work cannot be done.

The legs being covered, a left-hand glove is put on, unless one uses both—and the vulva is inspected. The left hand may suffice to reach both sides of the pelvic cavity, but in many

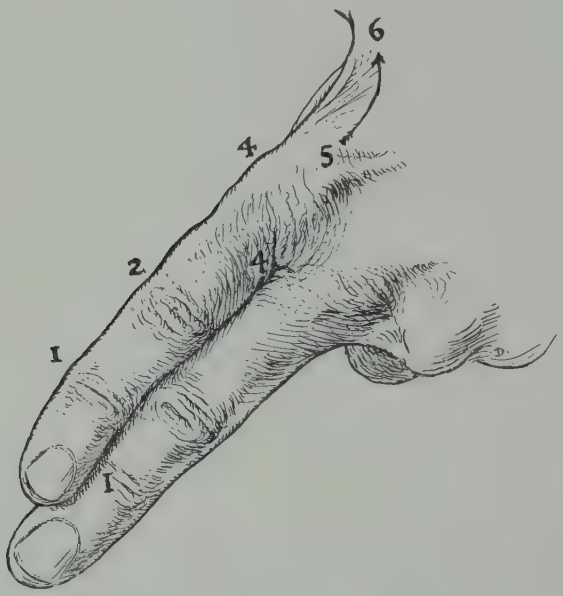


FIG. 11. Convenient figures on the fingers for laying off diagrams of tumors and displacements and alterations in these.

cases and in all difficult problems it will be desirable to change hands in order to palpate the entire right and left adnexal regions and make a complete sweep of the pelvis with the tips of the examining fingers. The upper hand pressing deep into the brim displaces the bowel with a rotating movement (Fig. 9); and then one causes the patient to breathe deeply and gets her attention off the lower abdomen if possible, in order to relax the muscles.

The rectoabdominal examination should be routine unless the vaginal walls are completely relaxed, and is the chief or

only reliance in the virgin or wherever there is tenderness or spasm at the vaginal opening.

In using the bivalve speculum, the smallest available size is to be chosen. All models but mine have the pivot, not located for the narrowest part of the passage, just within the hymen, but so far out as to pry open the lower vagina as well as its wide upper section. The speculum meets less resistance if it is placed while the patient is still in position with the hips elevated; then the table is lowered as the doctor sits down, in order to bring the cervix to the level of the eye. Where the ordinary bivalve is not effective, I find a long-bladed instrument of considerable value; and also had made a virgin size, distending the hymen no more than the index finger, with long blades and with the pivot at the hymen level so that the hymen is not distended by a wide separation of the tips. These, however, are unnecessary, because the only difficult vision —in the distance in a long or narrow vagina—is always feasible in the knee-chest or Sims' posture with good lighting by headlight or little electric bulb on a stalk. I treat most patients in the Sims' posture, because of the better vision of the whole undistorted cervix, the ballooning of the vagina, the accessibility of the upper part of the cervical canal, the readiness of dilatation of the internal os and the ease of placing of the tampon, whether dry or wet. The bivalve has a considerable field in Sims' posture, particularly when a man has no nurse. The bivalve is useful in the knee-chest posture to search for erosions in the posterior fornix produced by a pessary, or in placing the tampon. The knee-chest posture is

FIG. 12. Sims' posture, which is rarely depicted or taken correctly. Back projects slightly over edge; buttocks leave room to lay instrument on napkin; head is far to right; shoulder and knee close together; but above all, upper buttock is strongly displaced to right and away as compared with lower buttock: thus the direction of the vagina is downward toward table, its axis lengthwise of table.

the only one for firm tamponing. Take for example the woman with fibroids, menorrhagia and hemoglobin too low for operation. If one decides to tide her through one or more periods to better the blood color, the knee-chest posture is the only proper one for a firm tamponade. Two Sims' specula, a larger to lift the pelvic floor and a smaller to guard the anterior wall, furnish us proper machinery, since to be effective a tampon must plug every part of the cavity and give the maximal lift and drag upward on the cervix.

Sims' position is also useful in that it best exposes the anal region, while the knee-chest is the one for proctoscopy.

As part of the examination of the lower trunk, any woman complaining of backache must be studied for sacroiliac trouble. The leg, extended and relaxed, resting at the ankle on the doctor's hand, is swung up and down while the other hand palpates the joint and the patient reports whether pain at the proper spot is produced. The further test for backache is usually made after the vaginal examination or treatment, the patient, as she gets up, being asked to stand against a door jamb or door edge. This exhibits her attitude. If, the buttocks just touching, the shoulder blade is not more than two fingers' width away from the edge, she stands well and not in the forward or kangaroo position that puts constant strain on the back muscles. On the other hand, if she slouches in the gorilla position or if she sways back and forward, she has the weak back muscles that demand systematic exercises. Any of these three account for backache.

To complete the examination, particularly with backache, retroversion or prolapse, the patient is examined standing, after dressing, first for attitude against the door jamb, and then with a foot upon a stool. This final standing test will develop the extent of rectocele or cystocele, or will sometimes show that a uterus which when lying on the table is in partial retroversion, is in perfectly good position when the trunk is upright.

LEUCORRHEA

Most leucorrhoea has its origin in the cervix. The body of the uterus is very rarely at fault, and curetting for leucorrhea has been the subject of sweeping condemnation in recent years. Douching has its place, particularly until

treatment has produced a cure of the cause. However much has been written upon the subject, we constantly encounter defective technique in this simple matter. Women fail to pass the tube sufficiently deep or to lie down in using the douche, or to distend every fold of the passage by holding the parts together around the nozzle and then allowing the water to gush out. Therefore a printed slip of instructions should be handed to the patient when an order for the douche is given. A non-staining, non-poisonous, non-odorous preparation is to be preferred. Zinc sulphate or alum, one to two teaspoonfuls to the quart, is as good as anything.

Where douches fail, dry treatment sometimes succeeds. In the office the vagina is loosely packed with kaolin, which the Germans called terra alba. This is the white clay from which china is made and has not the dirty look of Fuller's earth. It should be washed out after a couple of days, as it may form an annoying cake or crust. The patient can apply it herself with a Nassauer powder blower three or four days running and then take a douche; then, after a day's interval, begin again with the powder. Very brief trial will demonstrate whether the patient is one of those to get well-defined relief from this measure.

ENDOTRACHELITIS

The largest section of office gynecology is the care of the inflamed or infected cervix. The most common of symptoms for which patients come to us is leucorrhea. The most frequent cause of sterility is shown to be cervical catarrh. Therefore more attention will be paid to this than to any other heading of the present chapter. The methods in vogue involve either frequent repetition of topical applications or else a repair operation or a removal of the diseased cervix. Where a granular eroded surface was rebellious to local applications, stitching was supposed to be necessary even though operations were done in each interval between several children. Both the prolongation of treatment and surgical relief have had their fields considerably narrowed by the use of the cautery. Two or three applications of the hot wire, spaced weeks apart in order to allow for rather complete cicatrization of the areas treated, will heal and inroll in a way that will

completely meet the need between babies, and will often render operation unnecessary. Furthermore, any measure that abbreviates office treatment and obviates the obsession of "womb trouble" that belongs to the chronic office habitué is to be welcomed. This method claims the large majority of chronic cervices—in between the simple raw areas that respond to a couple of applications of silver nitrate at one end and the extensive injuries that call for hospital work. The cases responding particularly to this treatment happen to be especially rebellious to others. These are:

1. Rough and extensive granulation, with eversion.
2. The cysts, superficial or deep (these recur sometimes after repairs).
3. Voluminous, adhesive mucous catarrh of the canal.
4. Gonorrheal free secretion with thickened lining.
5. Between-birth erosion with or without laceration (recurring with each labor if sewed in the intervals).
6. Patients whose physical condition precludes, or whose circumstances postpone, operation.
7. Marked endotrachelitis in virgins (because the visits are few).

There are groups that belong primarily to other methods, such as those responding promptly to simpler measures, or in women who cannot be subjected to any office treatment. These are catarrh with pinhole external os that calls for dilatation; chronic infiltration about the internal os requiring the dilator; edema, and edema simulating hypertrophy that need depletion; congestions due to constipation and sex-function disturbance; and endotrachelitis that is an expression of a general condition.

The cautery outfit that is most flexible and delicate is the one employed in the office of the nose and throat specialist. With the direct current the cost is about fifty (50) dollars; with the alternating current, eighty five (85) dollars. The generator which makes a vicious, spitting noise and is used with any current, alarms patients. Even the hum of the ordinary motor is feared. This should be done away with by hanging the motor in another room or in a closet, or better still, in the cellar, with a switch to start it, located close to the gynecologist's seat when at work. Of the several

platinum tips, the simplest and finest, as shown in the diagram, can do all the work, but a short spiral is effective for the rebellious catarrhs of the upper part of the canal. It should be noted that a low cherry-red heat is as effective as a white wire, and does not cause bleeding.

Fig. 13. Cautery treatment of endotrachelitis. The fine hot wire scores narrow gutters through granular areas (March); the remaining raw surfaces are again burned (Sept. and Nov.); shrinkage and inroll persist, and no operation is needed.

On the granular areas a tiny gutter is swiftly burned at ¼-inch intervals (Fig. 13). For small areas punctures are made. Repetition once in ten or fourteen days is the average, two or

three treatments sufficing. Progressive scar shortening does the inrolling later. For erosion with edema, shrinkage with boroglyceride can precede or accompany the cautery. Sticky secretion must be cleared away before the cautery is used. Narrow, rough gauze twisted in the canal does this best, such as the discarded cut-off edges of gauze squares, or inch bandage. The wide gonorrheal canal is treated with two stripes, the narrow by punctures.

We had been in the habit of treating obstinate adhesive catarrh behind a somewhat narrowed external os by stretch-

FIG. 14. Endotrachelitis. Obstinate catarrh of the cervix. Very large cavity with a narrow external os and rough gristly walls in the canal. Nasal cautery tip burns ditch on sides of canal.

ing the opening and using a tiny sharp curette in the canal (Craig or Delatour). The sensation imparted by the gristly lining of these long-standing cases is as if one were scratching over the back of linoleum or Brussels carpet. This is the very condition for which the delicate cautery strip works promptly. The canal is carefully dried out, the single tenaculum steadies it, and a longitudinal application is made on the two opposite sides of the wide oval passage (Fig. 14). If necessary sides not touched may be striped three weeks later. Neither undue contraction nor tender scars have resulted from these procedures.

The cystic cervix is especially adapted to cautery treatment (Fig. 15). There are some cervices so riddled with deep

cysts that only complete amputation will relieve the pressure ache. But even these are worth trying to cure with the fine-needle. Only, if one fails and desists, he must defer operation until the suppuration has entirely disappeared. The spear may cause a better depletion of a congested cervix, but its openings do not gape till granulation has closed the cavities,

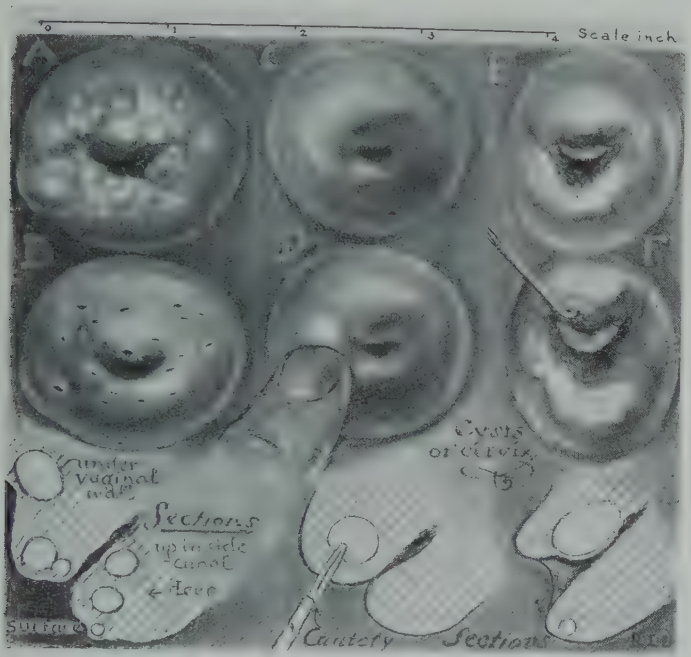


FIG. 15. Cyst of the cervix. A, before puncture, and B, a day after puncture with the delicate nasal cautery wire shown in the center at the bottom of the cut. C, large cyst bedded and invisible, made evident to the eye by D, finger pressure, ready to stab. E, cyst up canal demonstrated by eversion, F. The locations are shown in the sections below.

as is the case with the cautery punctures, and therefore the same pocket may heal over and fill again, if cleanly cut open by the knife. The cysts that the finger feels and the eye cannot find may be made visible by finger-tip pressure, quick withdrawal of the digit and a stab at the blanched spot that remains visible just long enough to reach. The tenaculum draws open the canal for interior Nabothian distentions. For

deep punctures the wire is to be very hot in order to keep on penetrating. In vascular and varicose womb-mouths the platinum should carry as little heat as possible—unless a decongesting ooze is desired. The amount of relief afforded by opening cysts is evidenced by the return of patients with cysts in new spots, voicing the request that the old ache be

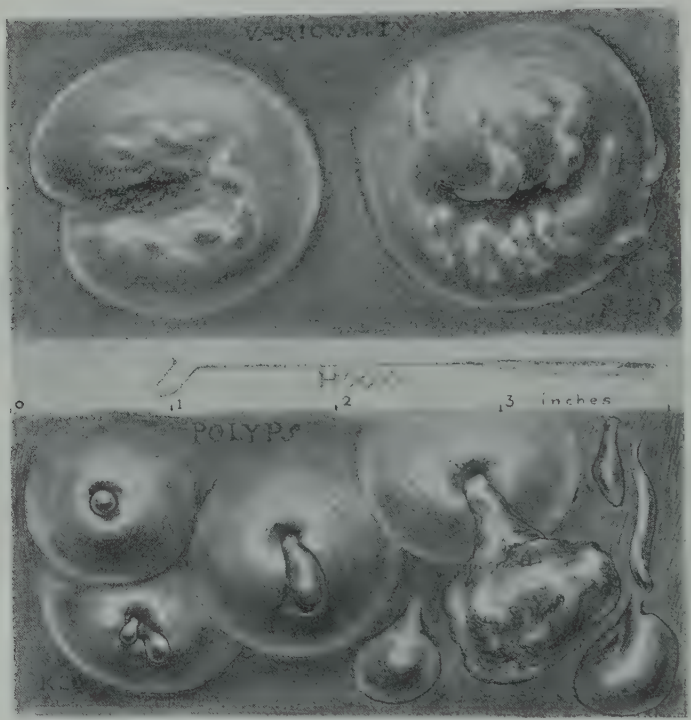


FIG. 16. Above, the rare varicosities, to be punctured by wire barely cherry-red; below, various polyps, to be lifted by the hook to reach the stalk with a bent wire cautery tip.

removed. They recognize the recurrence of tension. The puncture is curative, the intervals of recurrence lengthening from months to years. Cysts and cancer seem not to belong to the same cervix. Eversion is responsible for some erosion and irritation; the longitudinal cautery strips inrolls the surface and relieves certain cases.

Polyps are best handled with the fine cautery wire if the stalk is accessible. On the illustration (Fig. 16) there is shown an ordinary metal applicator bent in hook form, which conveniently lifts the stalk within reach of the tip of the hot wire. This should be passed up as far into the canal as one can reach along the stalk, with a low heat and with the utmost gentleness, lest free oozing begin and the little operation be obstructed by blood-flow.

In one-fourth to one-fifth of the instances the raw surface is acutely painful. Under these conditions a novocaine tablet dissolved in ten minims of adrenalin solution, 1–1000, packed into the cervix and left there for ten minutes after these surfaces have been thoroughly dried, is the best measure, but for quick action phenol will usually anesthetize sufficiently.

A second objection is cost and elaboration. It is true that argentic nitrate in 10–16 per cent strength has a wide range of efficacy, except in the cystic forms; that we can avulse polyps and that cysts can be punctured with the narrow eye-knife or with the Buttle spear. But the gynecologist cannot well dispense with the fine cautery tip for other purposes, such as urethral caruncle, chronically infected urethral glands, and the small hemorrhoid. Moreover, we have in the cautery the one enemy to cancer, should it have started in any part of the granular area; for silver nitrate is said to favor carcinomatous change if frequently applied.

After the external surfaces have been healed—or, in case of haste, at the same time with the external treatment—the deeper parts of the canal may be treated. These should be carefully wiped dry, and it should be remembered that a very little mucus will slow up the heating of the wire, which is out of sight in this high reach (Fig 14).

The treatment is particularly adapted to the endotrachelitis of the virgin, since it may be applied through a tiny Sims' speculum. The knee-chest position and one or two treatments will ordinarily suffice. Indeed, patients formerly requiring a plastic operation (and rupture of the hymen) are saved from operation in this simple fashion, and the unmarried woman is saved from a long course of office treatment.

This delicate procedure has supplanted the clumsy cautery method of the originator of the operation, for Hunner

advocated the thick Paquelin knife under anesthesia. It is simpler than the radium application of Curtis, is far less expensive, and has not the element of uncertainty which is attached in a certain degree to all uses of the gamma ray.[1]

The Tampon. The main use of the vaginal tampon is for depleting very marked congestion, as before a period or with considerable edema, or as a support after a painful treatment. Absorbent cotton is to be rejected, as it shrinks into a hard ball. The old type of cotton batting keeps a degree of elasticity, but is defective as a vehicle. Far better is wool, crinkled and long-fibered, if such can be obtained. This holds a large amount of glycerine, is elastic and will not mat into a hard pad. Ichthyol is a useless stench, no more effective than glycerine. One-tenth boroglyceride in a good grade of glycerine, the tampon carrying every drop that will not drip out of it, and backed by a second dry tampon, will produce a large amount of watery flow, sticky and uncomfortable, but effective in the few cases in which it is needed. Where marked benefit is experienced and continued treatment is desirable, the patient is given an order for the wool and for the boroglyceride, and allowed to treat herself with her own bivalve speculum; or she may use the wool-filled capsules on the market, adding glycerine.

The tampon should never be placed with the patient in the dorsal position, except with a widely relaxed vagina. The knee-chest is the position of choice, the Sims', allowable.

PESSARIES

To the pessary belong, of right, the retroversion of early pregnancy, the retroversions of the puerperium, the replaceable retroversions of women who are bad surgical risks. To this class belong also the prolapse of women who are very

[1] There is a cautery with silver points which is simple and fairly effective made by the Post Electric Co. at twenty-five (25) dollars that attaches to any light socket or wall receptacle. The "spear" point is the form most useful.

For teaching or demonstration of the cautery the cervix is modelled in plasticine and the wire merely warmed to cut into this clay-wax. Cysts are excellently simulated by burying gelatine capsules of various sizes at varying depths after filling with any jelly lubricant. They yield the fingertip findings and, on puncture, spurt mucus and gape. Whittled tomato or waxed Turkey red make polyps. Sections of cervix demonstrate treatments high up the canal. The student fashions his own models.

old, and those among the diabetic and nephritic which may be held in reasonable comfort by a support. Also, women of independent means with the invalid habit of life whose habit is incurable and in whom the risk or fear of operation is considerable may also justifiably be condemned to the perpetual pessary.

The cases to be let alone are, among retroversions, many,—among prolapses, none. The retroversions best let alone are those without symptoms or complications, those merely part of a general enteroptosis and those who symptoms are only evidence of neurasthenic sensitiveness. The conditions requiring pessary are:

1. Retroversion in early pregnancy (every woman to be examined; ring to be worn three months).
2. Retroversion in the postpartum months (present in one-fifth).
3. Retroversion between pregnancies while waiting until childbearing is past.
4. Retroversion in cases of relatively recent occurrence.
5. Retroversion in bad surgical risks.
6. Retroversion in extreme neurasthenia.
7. Retroversion in patients who cannot find time for operation.
8. Retroversion in patients waiting for operation.
9. After operation for retroversion.

Also in prolapse with the following:

1. Aged women.
2. Bad surgical risks.
3. Before operation, to reduce edema and heal ulcers.
4. In waiting for operation.

The conditions calling for trial of pessary are (1) Retroversion with symptoms but no complications;—e.g. dysmenorrhea and backache marked, but without tears or tumor; (2) Retroversion in some neurotics as a test; (3) To test whether symptoms are due to retroversion; (4) In some borderline cases; (5) To convince a patient that the pessary is not practicable; (6) In prolapse of the uterus alone, of slight degree and easily held.

Contraindications are lack of skill in use of pessaries; lack of opportunities to watch a patient with any pessary

which is not self-replacable by the patient; any degree of prolapse of the bladder which needs operation; inability on the part of a gynecologist with real skill to place a pessary that will hold the uterus in place after fair trial; persistent nervous disturbances after office treatments (mostly young virgins); some acute-angled retroflexions and, lastly, the presence of fixation or scant-range adhesion.

The selection of the pessary may be determined by measurement of the vagina, by experiments with various forms and sizes, also after standing, corseted. Soft rubber is to be employed only for trial or for self treatment by nightly removal. Hard rubber, or the equivalent, is always the choice. Small and medium sizes (three inches and under) fulfill the needs of 90 per cent of proper cases. A gradual reduction in size is to be carried through, and finally removal of all support. For nightly removal in prolapse use a circle or ball with handle (Menge, Dickinson); or a Hodge with scant S curve. Few forms and few sizes are needed for retroversion— the Smith and Hodge, an occasional soft rubber ring or the Thomas thick upper bar. The Gehrung sledge holds a few cystoceles, and intelligent patients can place it.

As a precautionary measure in watching the pessary, printed directions giving details of the biweekly douche, and for regular return to office, are given. Weekly examination is directed at first,— monthly or bimonthly later. The inspection is for chafing or ulcerating of the vagina, or crusting of the pessary. An entry is made on the diary or a tab placed on the history to flag pessary cases for recall to the office. A test by removal after three or six months decides whether the pessary is needed any longer.

STERILITY

Sterility and non-operative treatment have an intimate correlation. We now know that nearly one-half the fault lies with the male partner; but it often falls to the gynecologist to examine semen, because it is the woman who is most interested in most cases, and who will take the necessary steps to send the husband. Moreover, it saves the expense of a consultation with an urologist and keeps the study of the puzzle all in one's own hands. Furthermore, one of the

important tests, the search for live or dead spermatozoa in the cervical canal (Huhner), belongs to our department. Where the semen in the unchilled condom, or self-produced in the office toilet, is found normal in forms, active, not clogged with mucus and showing two to four hours' vitality at room temperature, or where the forms found in the vagina or cervix after coitus are normal and moderately active, we proceed with the study of the woman, and can offer her one chance in four for a cure, unless she has closed tubes. One may not too strongly condemn either treatment or operation undertaken before the character of the semen is determined.[1]

The male being exonerated, the patency of the tubes is usually the next question. This test is only to be made with the cervical canal reasonably healthy (so that infection may not be carried into the peritoneal cavity) with the urethral glands uninfected, and in the absence of swelling and tenderness suggesting any degree of active tubal trouble. Happily the expensive and complicated Rubin outfit with its valves and meters is unnecessary. The uterine tube connected by a T with the ordinary pressure gauge of the sphygmomanometer and with a bulb obtains the evidence (Fig. 17). As a test it suffices that gas goes through. The amount and rate are immaterial. The posture may be dorsal if the uterus is in partial retroversion, but if anteflexed or, in my opinion, if in the usual position, even if mobile, Sims' position or the knee-chest should be used. If the canal is free from mucus, a wipe of tincture of iodine suffices; if there is clear mucus in any quantity, it should be cleaned away before the iodine. The anterior lip is grasped with a double tenaculum like the Skene, with slender, over-lapping sharp points that are less painful than the blunt, short prongs of the bullet forceps. The tip of the uterine tube should pass the internal os, because this portal may sometimes yield a spasm resistance of 200 mm. Before introduction a gob of lubricant is smeared on the shoulder of the tube so that bubbles passing backward will be demonstrated. The shoulder is driven hard against the external os, hard enough to resist the maximum pressure used. The doctor watches the cervix to detect regurgitation,

[1] When the husband fails to keep his promise to appear, the woman may catch the semen in a bottle immediately after coitus, and bring it to the office, with minimum delay and no chilling.

while the nurse keeps her attention on the meter; but the meter can be hung close to the vulva, so that it can be watched out of the tail of the doctor's eye, though this is

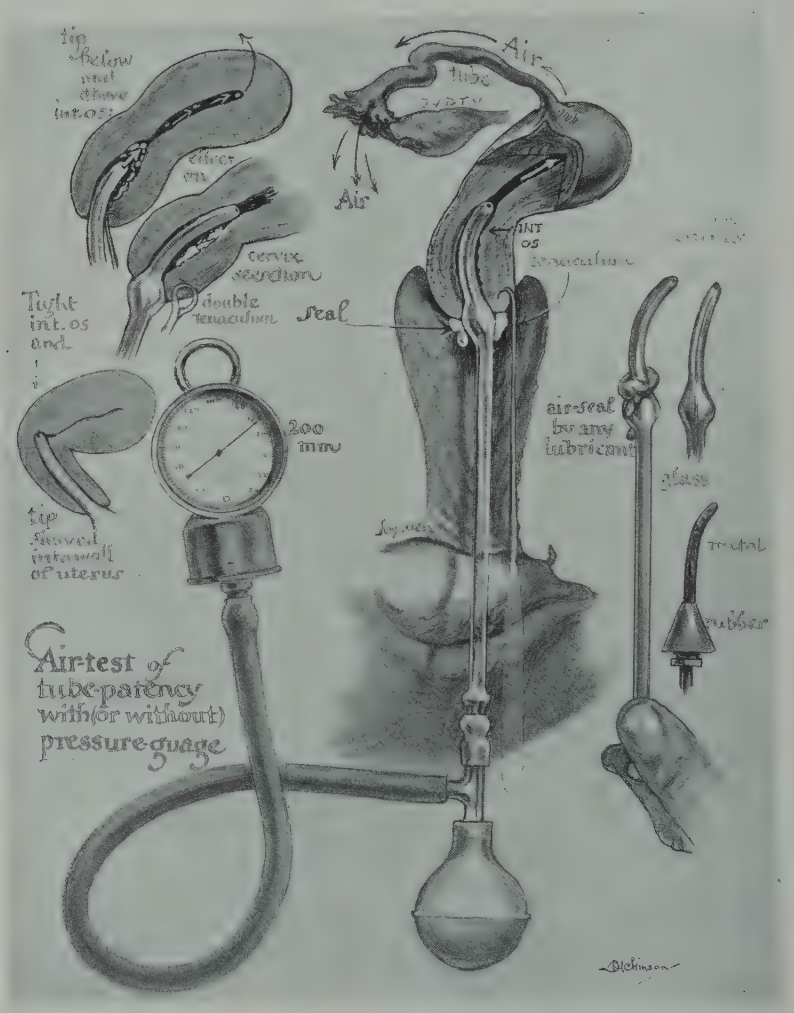


Fig. 17.

less accurate than double observation. The nurse calls off the figures as the needle rises. Rate of inflation has not seemed to me important, but it is of value, on reaching the

higher figures, to hold on steadily in order to give from a quarter to a half minute for any spasm to subside.

The test is made half-way between the periods, that is, at the time of least congestion of the lining. It should be repeated in a month, before a decision is given. One tests up to 200 mm. From 40 to 80 mm. is an average resistance of open tubes; from 120 to 180 mm. the tubes are kinked or otherwise partly occluded, as in retroversion. When partly occluded, subsequent insufflations may show yielding at lower pressures, but the number of pregnancies following attempts to open up tubes that allow passage at 150 mm. and over has not been many. In other words, up to this time insufflation has been of incalculable use in diagnosis, but not of much promise as treatment.

Among causes of sterility, Polak has shown the leading place to be taken by endotrachelitis.[1] I have demonstrated that a modified Huhner cautery is the most effective remedy for this disability. Treatment of catarrhal conditions of the cervix and of cervical stenosis is the most valuable combination. The cures accredited to curetting in the past, we now believe to be largely the results of dilatation. This dilatation of the external os, giving freer drainage, and of the internal os, overcoming spasm, is a proper office treatment. As compared with the graded Hegar sounds, the slightly curved dilator of Palmer and the strongly curved dilator of Goodell, that adapts itself to the canal of anteflexion, give the maximum stretch and minimum distress, because the act is all over in a second.

Anteflexion as a cause is stenosis or stenosis plus catarrh. Retroversion as a cause has recently been shown to be largely complicated with occlusion of the tubes,[2] even not adherent; but adhesions are to be expected in at least one-third of the cases, and endotrachelitis may be the real cause in another third of the cases of retroversion. Reposition and the pessary

[1] Huhner, Guy L. and Wharton, Lawrence R. Sterility: a study based on a series of 526 patients (*South. M. J.*, April, 1924, xvii, 269–277) found one-third with cervical discharges, one-seventh with acid cervical leucorrhea; and 157 women with cervical catarrhs who had sound husbands showed 25 per cent cured, chiefly by the cautery.

[2] Aldridge. Insufflation of the uterus and fallopian tubes, 600 cases. *Am. J Obst. & Gynec.*, July, 1923, 6, 1, 53.

may unkink the tubes and give the average percentage of cures, 25 per cent. The sterility due to adherent retroversion is incurable without operation and then rarely.

Tubal Infections. I have found a history of previous appendicitis in a considerable number of patients, but the usual cause of closure of the tubes is gonorrhea or the lighting up of a low-grade infection following delivery, abortion or curetting. This is not the place for the discussion of laparotomy for adherent tubes. Suffice it to say that its main advocates, Childs and C. C. Norris, have published their successes but have not stated their percentage of failures.[1]

Artificial insemination must be restudied in the light of tubal patency tests, as we now know that it was absurd to attempt these injections with the 30 to 45 per cent of closed tubes found in sterile patients. Only good semen should be used. The opening of the pipette should not be minute—else the passage through the narrow opening kills the sperms—and the period of trial should be between the sixth and the twelfth day following the beginning of menstruation.[2]

Personally I have seen no benefit from treatment of sterilty with *animal extracts.* The attack of epidemic endocrinitis from which the profession has been suffering shows some subsidence. No results have been credited by me to calcium lactate, nor to forced nutrition. With high degrees of vaginal acidity the soda douche, two teaspoonfuls to the quart, has produced a few successes when regularly practiced within an hour before intercourse.

Sterilization in the Office. The procedure consists in the production of a burn that brings about a slough and thereby an impermeable circular scar stricture at the point where the Fallopian tube enters the uterine cavity, at a spot where the canal is only of the size of a bristle. A cautery tip on a sound does this, or the ordinary nasal electrode (Fig. 18).

[1] Huhner had three pregnancies following twelve tubal operations, eight of them being salpingostomies and three, freeing of adhesions. (One-seventh of Huhner's total cases treated were cervical stenosis, with 33 per cent cures.)

[2] Seigel, P. W. (1) Wann ist der Beischlaf befruchtigend? *Deutsche med. Wchnschr.* Freiburg, 1915, No. 42. (2) Bedeutung der Kohabitationstermines f. d. Befruchtungsfaehigkeit der Frau. *München. med. Wchnschr.*, 61, No. 21.

The field lies in cases where there is no indication for opening the abdomen.

Unless the uterus is in partial or considerable retroversion, Sims' posture is to be preferred. The cervical canal is to be

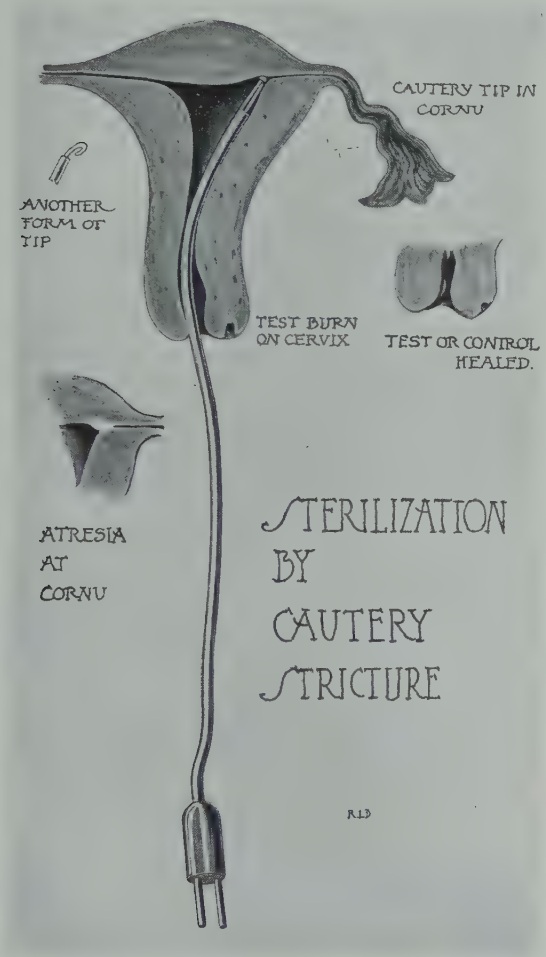


Fig. 18.

wiped clear of mucus and the time chosen when the endometrium should not ooze—half-way between the periods. Local anesthesia can be done by 5 minims of adrenalin solu-

tion of novacain, introduced with low pressure (20 mm.) through a pipette, allowing ten minutes for the effect. Then the cervical canal is tested to see that the internal os is sufficiently open and the sound passes on to study the shape of the body of the uterus and the distance from the external os to the cornua. This distance is noted and, on one form of cautery sound, the slide is run up to register the same distance on its shaft. The cautery sound is tested for a moderate heat. In one's earlier work one provides a control burn by a preliminary application of the heated tip to the cervix or the open canal. This indicates the minimum number of seconds needed to bury the platinum—say four to eight, according to the heat. Within the cavity a longer time will be needed, in case the wire is clogged with mucus or blood. Now the cautery sound is passed up to the cornu and the current switched on. In removing, there should be well-defined resistance, which gauges the effect. There is no fear of perforation as it has been shown to take over twenty seconds to penetrate the wall of the uterus. The second side may be done at another time. The pain is not severe, even with no local anesthetic. Some staining may ensue for a few days, and the following period be troublesome.

Insufflation to test the success of the occlusion is to be deferred for at least two months to give time for contracture. The earlier closures antedated insufflation and were not proved, but of eight tested strictures the two failures are in a lopsided cavity with only one palpable horn, and in a uterus with an exit pressure of 20 to 30 mm. The others stand 200 mm., though previous to cautery closure they ran from 60 to 105 mm.

IRRITABILITY OF THE BLADDER

A very large proportion of our office patients complain of frequency or urgency in urination. Theoretically a cystoscopy should be done in all such instances for complete diagnosis. However, as almost all of them exhibit a form of trouble yielding readily to simple treatment, such treatment furnishes an ample diagnostic index. Trigonitis and urethritis of the upper fourth of the canal, with or without edema and stricture, may be expected to be present without any other

lesions. The treatment is simple. Applications to the completely emptied bladder, and stretching of the upper urethra suffice. The stretch may be done with a small dilator in case the applicator itself does not suffice to give the necessary widening. One is surprised to see the large degree of relief resulting from so simple a procedure. The two-way catheter here pictured is nothing new in principle, but it has the con-

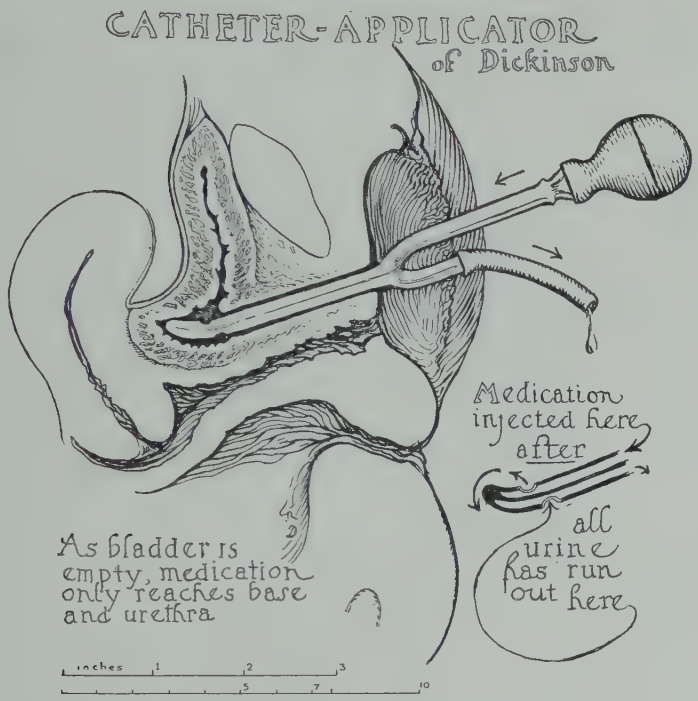

CATHETER-APPLICATOR
of Dickinson
Medication injected here after
all urine has run out here
As bladder is empty, medication only reaches base and urethra
inches

FIG. 19.

veniences that insure visible cleanliness, a shoulder which prevents too deep an introduction, and, above all, it allows of the drainage of the last few drops of urine through the lower half before the medicament is injected. Thereby the solution used is not diluted, or, in the case of nitrate of silver, precipitated. As an application my own preference is for 10 per cent protargol. The frequency of use runs from once a week to every other day, according to the amount of relief

afforded. I teach intelligent patients, living at a distance, to treat themselves.

The dilator is a miniature glove-stretcher, built on the principle that the area requiring stretching is far up the canal, and that stretching of the narrowest and least flexible part

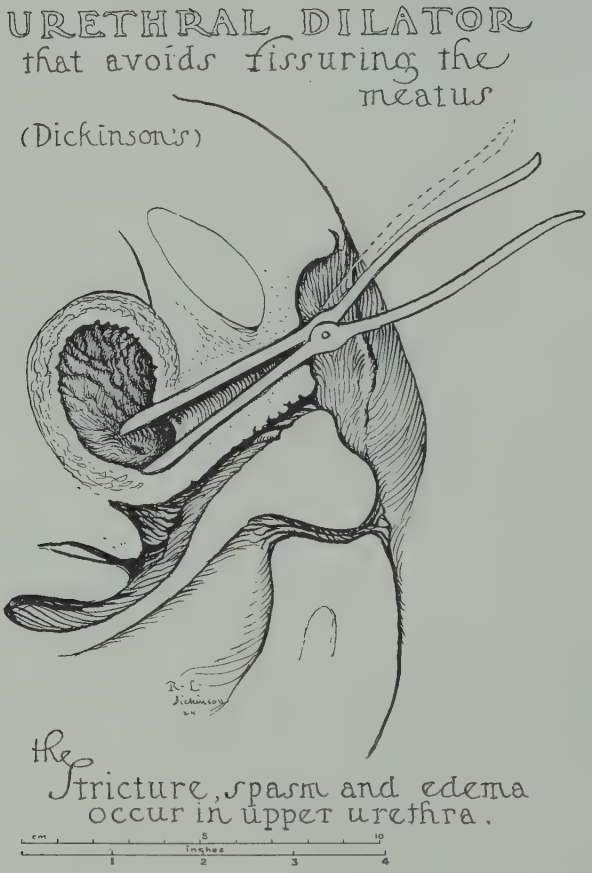


Fig. 20.

of the canal, the meatus, produces fissure and very painful urination.

Any doctor who is doing much gynecology can readily familiarize himself with the use of the diagnostic cystoscope, provided he uses the child's size. This, being no larger than a

slate pencil, enters with ease, gives a good view of the ureteral openings and of the bladder base, exhibits ulcers or determines the absence of a growth or demonstrates pus coming from one of the ureters—in other words, will determine, in connection with the history, whether there be any gross lesion probably present that would call for the services of an urologist.

RECTAL INSPECTIONS

No gynecologist can presume to make a diagnosis in the matters of pelvic pain and backache without being able to conduct simple examinations of the lower end of the bowel. Inasmuch as one-half of our office patients are said to be constipated, and nearly all of our most nervous patients suffer from colitis, and ovarian, tubal and parametric troubles which are frequently complicated with rectal and sigmoid congestions, it behooves us to make a routine of visualizing the rectal mucosa. This is entirely simple in the knee-chest position with the cheap tubular speculum of Kelly and the head-mirror, or with the light on the slender stalk that we need in using the tiniest virgin speculum. Thus we may find an extreme degree of spasm; swelling sufficiently acute to bleed at a touch; large quantities of mucus; hard masses blocked above the third valve, even after a movement, or ulceration; inflamed anal tabs, or the patchy mosaic of chronic proctitis. Here again very simple measures like irrigations or oil enemas will yield us an amount of credit out of all proportion to the labor involved—credit due merely to thoroughness in diagnosis.

WRITTEN DIAGNOSIS GIVEN TO PATIENT

A patient or the family is entitled to an opinion in writing. To give no more than a verbal statement is not to deliver the goods for the money, inasmuch as the general ignorance concerning anatomy and disease is such that errors in understanding and emphasis are otherwise unavoidable. In simple English, with the Latin sometimes in brackets, the findings and suggested treatment can be briefly phrased. It is only in certain findings of peculiar secrecy or on a point-blank decision such as "pregnant" or "not pregnant" that a written statement is superfluous. Even then the carbon constitutes

evidence occasionally desirable later. It does call for courage to put all our conclusions in writing, aware as we are of the wide element of error in medical and surgical diagnosis. At the same time I have found that where the diagnosis has a doubtful factor, entry to that effect saves face at some future time. Beyond this one may go, by a memorandum, to the effect that it is to be understood that the diagnosis or advice is based on the findings of the present date and the amount of study so far made, and that further facts may be forthcoming with longer investigations. At any rate intelligent people appreciate the report, and there are instances of a kind in which such omission will not be condoned in the future, as in the matter of a summary of the findings and steps of operation to be rendered to the patient as her simple right, at her last office call.

OFFICE INSTRUCTION IN SEX HYGIENE

When giving the foregoing "practical talk" there was a deliberate omission of a subject that constitutes an important section of office gynecology. It is a difficult duty which may not be shirked, for the reason that there is no one but the doctor qualified to undertake it. It begins with the offer of advice under suitable circumstances, and works out into a more or less set speech on each of the following subjects—my own formulas developed from twenty years of trial, and from requests for subsequent criticism. (1) Hints to mothers on how to approach autoerotism with their daughters; (2) discovery of those unmarried patients who have a fear of the physical aspects of childbirth or marriage, and removal of these inhibitions; (3) carefully worded warning to a girl likely to have a long engagement, and to her fiancé; (4) offer of free advice to the prospective bride and groom just before marriage, in separate interviews; (5) instruction on the hygiene of marriage, on the unwisdom of deferring the first child too long, and on the technique of contraception—this knowledge on the matter of the proper and voluntary spacing of children being furnished in all cases except where one fears the information will be abused for selfish reasons; and lastly, (6) after each birth, advice as to the best time for the next arrival, and data as to the way to adjust the arrival.

Some day the specialist society will assume its obligation to provide some kind of standard of instruction in this matter. Meanwhile a lecture to senior medical classes has been given a trial, with a summary or outline prepared for them. In any case, this aspect of office gynecology can no longer be allowed to go by default and be left to the laity, for lack of courage to face a distasteful responsibility.

In conclusion, this scribe desires to say that, in bringing forth out of these measures a few things new and old, he asks leniency for his attempt to compress the material of a book into a single chapter.

RATIONAL PATHOLOGY AND THE NEW THERAPEUTICS*

JAMES EWING, M.D.

I AM asking your attention to a topic in medical philosophy. The most important thing about a man is his philosophy. If that is true of a man it is also true of medicine, for whither we are headed there we are bound to go. It is well worth while that we should examine the tendencies of modern medicine to see whether they are sound.

One of the most conspicuous tendencies of recent medicine is the high development and constant use of laboratory methods of diagnosis as well as the quantitative estimation of the phenomena of disease, and the practice of venturesome therapeutics suggested by these methods; while at the same time there has been relative neglect to cultivate the older and more substantial and direct methods of study and diagnosis, and to practice conservative therapeutics. This tendency, I think, should be submitted to careful scrutiny.

The first and probably the most important body of substantial information on which practical medicine is based, is morphology, pathological anatomy and histology. Failure to recognize this fact has disturbed the proper development of medical education and the progress of medical research, interfered with hospital organization, and increased the distrust of the public for the medical profession. Now, of course, I do not expect to prove all these statements to all of you, but shall be satisfied if some of you become impressed with whatever truth may be contained in them.

You will recall that in the beginning of the nineteenth century great changes took place in the conceptions of medicine. When pathological anatomists did away with the chemical conceptions of Galen's time, they introduced the

*This lecture was delivered on Oct. 13, 1922.

idea that for every disease and every functional disturbance there must be an alteration in structure. Then began the era of pathological anatomy on which modern medicine of today may be said to rest.

Everyone knows what a radical transformation was brought about by the old school of European pathological anatomists when it was insisted that speculation should cease and demonstration of morphological changes in the organs should be made before accepting the conceptions of disease. Yet the day of pathological anatomy has passed. It has done its main work, and if we are to make progress in the future we must pass beyond this field and develop other finer and more accurate sciences. So today, in looking over the field of medical research, one would say that bacteriology deserves consideration over morphology.

Bacteriology has been, perhaps, the most fruitful of all branches of medical science. Within a space of about fifty years it enabled us to diagnose, and somewhat successfully treat, most of the infectious diseases. The germ theory appeared at a most opportune period in the history of medicine. It has made the earth habitable, and it has developed the arts. A knowledge of bacteriology is of the highest importance to the practitioner, but the greatest achievements of bacteriology lie in the field of preventive medicine, in which it is being slowly replaced by a larger science, epidemiology.

No one would for a moment depreciate the importance of bacteriology. Its importance is generally underestimated. And yet there are limitations to the significance of purely bacteriological knowledge of disease. The old morphologists believed that bacteriology could never give a complete explanation of disease, a view which receives increasing support in recent times. Modern bacteriological research is getting away from the study of bacteria themselves, and turning more and more to the questions of predisposing and contributing causes of disease. In other words, it is reverting to the field of general pathology.

Technical bacteriology has grown old very rapidly, one may say. It has children of the second and even third generations, in such specialties as immunology and serology,

which are very progressive branches of science. Yet students in these fields of research are showing some signs of fatigue. Only two years ago the American Society of Immunologists agreed to merge its meetings with those of the Society of Pathologists and Bacteriologists, a compromise which they would have scouted five years ago. The acute interest in immunology and serology is not quite so intense as it was five years ago. When bacteriology takes refuge in almost invisible filterable viruses, it comes to a dead standstill, as in influenza and poliomyelitis. So far as I know, we make practical use of only one filterable virus.

Bacteriology has contributed in very large measure to the progress of medicine, but it furnishes only partial knowledge of disease. We must know more about contributing and predisposing causes before we can explain disease.

Of all the branches of medical knowledge pursued today, chemistry undoubtedly is the most popular. If we interpret chemistry in a broad sense, this may be called the age of medical chemistry. Of course, in all departments of medicine there is a chemical side. Chemical investigation of disease has sharpened and clarified our knowledge. The main body of our recent knowledge of certain diseases, such as diabetes and Bright's disease, is chemical. Yet in neither of these diseases has chemical investigation furnished a practical means of control. After many years of waiting, the insistent demands of morphological study of the pancreas are met by a young chemist, who concentrates and purifies insulin.

Chemistry has one strong attraction to the medical mind, in that it offers exact knowledge, and we want exact knowledge in medicine. However, assuming that it is exact knowledge, it does not necessarily follow that it is important knowledge of disease; and from my point of view, as I peruse chemical contributions and read chemical literature in medicine, I am impressed with the feeling that its contributions and its aims are mostly concerned with the secondary phenomena of disease, and not with the primary factors. You may get from your laboratory a report that the non-protein nitrogen of the blood is very high, but that does not tell you whether the patient is suffering from Bright's disease, or a tumor of the ureter or of the bladder which

is obstructing the flow of urine, or from ascending suppurative nephritis, or from any one of a dozen causes which prevent or interfere with the excretory function of the kidney. In blood chemistry you are dealing with secondary phenomena. The important thing is to know from which one of the various causes of these secondary phenomena your patient is suffering.

Consider what a great amount of labor has been and is being spent on the chemistry of rickets. Calcium metabolism is undoubtedly disturbed in this disease, and fat-soluble vitamin is an important ingredient of diet, but it appears rather clearly that the cause of rickets is lack of fresh air, sunshine and exercise.

What a wonderful field for chemical and physiological study is Addison's disease; and yet the important factors in this incurable malady are the tubercle bacillus and status lymphaticus. So, to repeat, chemical knowledge of disease relates, as a rule, to secondary and not to primary phenomena.

Chemistry is a very attractive pursuit for the young clinician. It is always a new study. A talking knowledge of chemistry may enable a young man who has comparatively little knowledge of general medicine to make quite an impression. He can complete a study of an interesting phase of disease in a short time and acquire considerable reputation based upon his completed chemical studies. It has been said recently that chemistry makes great physicians very rapidly. There are limitations to the value of a knowledge of the chemistry of disease which I think will prevent it from ever becoming a final solution of the real problems of many diseases in which we are interested.

I think too large a measure of the resources of practitioners, of hospitals, and of educational institutions is at present being devoted to the pursuit of the chemical phases of disease, which takes the place of matters which are more important. This work belongs more properly to research institutions than to hospitals where consulting practitioners are being trained.

Recently a group of eight distinguished chemists published a brochure with the object of improving the position of

chemistry in medical education and research. They set forth the achievements of chemistry, all of which we acknowledge, but they made promises, which I think are open to question, that the cure of certain diseases would in time be furnished by chemical research. They suggest that the cure of pneumonia, and even of cancer, will surely come from the chemical laboratory. They set forth amyl nitrite as a cure for angina pectoris, and thyroxin for myxedema. They criticize the organization of research institutions because they are not largely chemical, and they denounce the plans of educational institutions because chemistry does not take a larger share of the curriculum. At the same time they have resorted to the publishing of propaganda in the daily press, which some of you may have seen. They claim that chemistry should take the leading place in medical research. They hint to the public that the chemist is a physician. They fail to remember that chemical conceptions held sway for nearly a thousand years at a time when medicine made very little progress. To my mind it would be unfortunate if the chemical conceptions of disease were made any more prominent than they are at the present time.

Chemical investigation is indispensable in the pursuit of many problems of disease, and this type of research deserves the abundant support which it now receives, but an intimate knowledge of medical chemistry is far less important for the practitioner than a knowledge of bacteriology and pathological anatomy.

Now, why is general pathology, and especially pathological anatomy, so important for practical medicine? Why must the general practitioner, the surgeon and the physician rely upon a knowledge of this subject rather than upon others? The first essential reason is because man is a bulky animal. He weighs 100 to 200 pounds. He suffers from the effects of gravity. For him the most effective therapeutic measure is rest in the horizontal position. He consumes prodigious quantities of food and drink, from which he excretes a bulky waste through a long tortuous intestine. Several feet separate his peripheral from his central nervous systems. He is a big machine, and it usually takes a considerable injury to interfere seriously with his well-being,

and especially to destroy him. Of course, he has certain points of particular vulnerability, notably his central nervous system. As a rule, man does not suffer from disease, and does not have very serious disease without gross structural changes in the body, the recognition and control of which mean the successful treatment of the disease. We await the exanthemata in the infectious diseases, the consolidation of the lung before we recognize pneumonia, and we detect anatomical changes in the organs in practically every instance when we venture to make a positive diagnosis and recognize a definite pathological condition in the body. A very important branch of medicine—surgery—is mainly concerned with the correction of gross abnormalities in the body, evacuating pus, removing limbs or organs, correcting deformities, sidetracking channels, relieving pressure upon hollow organs, etc., etc., and the instrument used, as a rule, is the sharp scalpel. The surgeon may bring his patient to the operating table with an increasing number of analyses relating to the secondary phenomena of the disease in question, but he will never be free from the necessity of wielding a definite powerful instrument—the sharp knife, guided by the trained mind. So that all these branches of practical medicine act most directly when they have at their disposal an accurate knowledge of the gross changes in the organs in disease. Lacking this knowledge, surgeons not infrequently say: "There is pathology in the gall-bladder," or "There is a malignancy in the breast," using these abstract conceptions instead of specific terms to cloak their ignorance of the exact conditions they have to deal with.

X-ray diagnosis is a field which has developed to a surprising extent. It is nothing more than a study in pathological anatomy, and the interpretations of the radiologist are reliable only so far as he visualizes gross changes in the organs. That is why radiologists always spend several years of study in the deadhouse.

A very sharp critic, Mackenzie, has taken the whole scheme of modern medicine to task, especially the pathologists, against whom he is most severe, saying that we do not get any of the pathological changes until it is too late. He holds that the thing to do is to study disease before

structural changes take place and when disease is remediable. This is a very important idea. Much more can be accomplished by careful study of the clinical conditions under which disease develops. Yet I do not believe society will, in our time, see the realization of this Utopian scheme. If it did, half the population would be devoted to taking care of the other half. Most diseases will always be encountered after they have produced definite structural changes in the body. In fact, it is probable that an alteration of structure must be included in the definition of disease. Otherwise every passing disturbance of function becomes a disease. The average man cannot stop because of minor symptoms of disease. He has to work on until he is incapacitated. We cannot, as physicians, devote ourselves extensively to the beginnings of disease. We do not do any too well in recognizing and treating established disease.

The great importance of pathological anatomy in diagnosis may be illustrated by referring to some concrete instances in which the failure of the study of this field has worked against the interests of physician and patient. Such instances are of daily occurrence. Some years ago a distinguished physician suffered with an acute febrile attack with signs of consolidation in the lung, in the course of recurrent carcinoma of the neck. He was in a serious condition and his eminence called for a consultation of the leading men of the city, ten of them in all. After consultation they concluded he was suffering from a bulky metastasis in the lung, although squamous carcinoma never produces bulky metastasis in the lung. We often see well-trained and capable surgeons industriously palpate lymph nodes in the neighborhood of a rodent ulcer, although this disease never involves the lymph nodes.

Not long ago a colleague of mine demonstrated 3 cases of lobar pneumonia caused by emboli from varicose veins in the leg. What would be the value of determining the types of pneumococci in these cases? Fat embolism, a very important cause of serious disturbance following operation, has had to be rediscovered in recent years, although the data known about it are in the literature of many years ago.

We love to indulge in very abstruse discussions of the nature of malignancy and the way in which tumor cells become loosened and find entrance into the blood and are carried to other parts of the body; when, as a matter of fact, very often the means by which such tumor cells are loosened from the original tumor is repeated examinations by the surgeon, the patient, and the patient's friends. One could multiply at length instances of this sort where a lack of knowledge of pathological anatomy has rendered the clinician unsafe in his diagnosis and therapy.

But what place has pathological anatomy in modern research? It has a very important place. I am constantly impressed by the feeling that modern research suffers greatly by the neglect of pathological anatomy. Not long ago a distinguished pediatrist reviewed the recent studies of marasmus in children. A number of leading pediatrists, here and abroad, have devoted themselves to a study of marasmus and have pursued several modern theories, mainly chemical. Finally, at the end of a series of studies systematically carried out in various countries, it is concluded that marasmus is due to an abstraction of water from the infant's body, a conclusion which could be told them by any pathological anatomist who ever performed an autopsy on the body of a marasmic infant. No explanation was given as to how the water was abstracted and how the infant's body can be enabled to absorb the water and keep up a normal balance. It added nothing to our knowledge except very detailed information about the secondary phenomena of marasmus.

When anaphylaxis was first discovered, the sudden death of guinea pigs which had been sensitized by the injection of proteins receiving a toxic dose, was a very puzzling phenomenon. It took the experimental pathologists and immunologists four years to reach the conclusion that the animals died from asphyxia, a conclusion easily reached by the pathological anatomist who is accustomed to see subjects dead from acute asphyxia.

A lack of knowledge of ordinary gross pathology, it seems to me, greatly retards the progress of experimental research in many fields.

There is one unique advantage of a knowledge of general pathology in the very broad generalizations which studies of this sort permit us to make. Bacteriological observers have noted that certain infectious diseases kill with great rapidity. A group of old-fashioned pathological anatomists in Vienna, headed by Paltauf and Kolisko, observing these phenomena, formulated the doctrine of status lymphaticus and forced its acceptance over a wide territory in medicine. How else could this generalization have been conceived? What did the bacteriologist or the chemist contribute to it? They may now, however, safely engage in its study.

The French doctrine of neurofibromatosis is equally extensive in its ramifications. It shows how subjects of a congenital disturbance of the fetal ectoderm suffer and must suffer from a wide variety of diseases.

These two examples are sufficient to show some of the advantages of this method of study. Who can doubt that in the future some broadly trained observer with a wide experience in clinical medicine and the modern sciences will be able to draw equally broad and significant generalizations?

When we come to add the microscope to the study of pathological morphology, we at once greatly broaden its field. I do not need to defend the microscope. Everyone knows its effect on the accuracy of our knowledge of disease. Even today, when it is used as a matter of course, it exerts an almost tyrannical influence in many fields, because it is so decisive, and to the well-trained eye looking through the microscope, it is a method of very great accuracy.

A well-known chemist some years ago denounced pathological histology with pathological anatomy as a dead science. He said that pathology is looking at dead cells through a brass tube. That is what it looks like; but a great deal depends on who is looking through the brass tube, and what information he brings to interpret the changes under the microscope. The great advantage of morphological interpretation is that it deals, as a rule, with the primary phenomena of disease.

What is the use of offering these illustrations to emphasize the importance of morphology in modern medicine? I sup-

pose all of you are more or less in agreement with me that pathological anatomy is the basis of diagnosis. But why should it stand in the way of the development of the other sciences? I have no idea whatever of recommending that studies in the known facts about disease should in any way interfere with the development of the modern medical sciences. My object in bringing this matter before you is a practical one. As originally said in my thesis, I believe too little attention is paid to this branch of medicine. We do not adequately provide for it in our hospitals, in private practice and in medical educational institutions. I believe that neglect of this important branch of medicine has been responsible for many mistakes in diagnosis and for most of the loose therapeutics of the present day.

I am supported in these views by a very distinguished authority. Strumpel, of Berlin,[1] has recently reviewed the state of modern therapeutics in Germany and compared it with the conditions existing thirty years ago. You will probably agree with me that no one is better equipped than Strumpel to form a correct judgment of modern therapeutics. He divides it into four classes:

1. Necessary therapeutics, such as the removal of the appendix, or the opening of an abscess, or the use of other urgent surgical procedures; or the giving of quinine in malaria and mercury in syphilis.

2. Useful therapeutics. A very large part of the work of the physician is giving useful drugs that relieve symptoms, but do not cure.

3. Useless therapeutics, where the procedures employed have no definite object in mind, and no definite accomplishments.

4. Noxious therapeutics. He refers to two fields of noxious therapeutics. He deplores the great amount of harm done in Germany with salvarsan administered on the assumption that if a man gives a positive Wassermann it means syphilis, and he must be treated with salvarsan with large and repeated doses. We are perhaps all familiar with some of the results of this indirect method in the diagnosis of a very important disease. Most of us are now aware of some of the

[1] STRUMPEL. *Deutsche med. Wchnschr.*, 1922, xlviii, No. 1.

dangers of salvarsan therapy. In another connection[1] he states that salvarsan often acts as a provocative of syphilitic injury of the nervous system, and that early syphilitic lesions of the nervous system, formerly rare, are now frequently encountered in cases treated with salvarsan.

He distrusts the psychoanalysts. I am not competent to speak of this new branch of medicine. I have heard it roundly denounced, and have also heard it highly praised. Strumpel thinks it does more harm than good in Germany. Strumpel's analysis of modern therapeutics is an authoritative and accurate estimate, I imagine, of some of the changes that are going on in the practice of medicine.

I am personally considerably impressed by the new type of dead-house pathology we see, illustrating the dangers of modern therapeutics, which we did not see twenty-five or thirty years ago. Operative deaths following the cholecystectomy are shockingly frequent. The surgical diagnosis and treatment of brain tumors has not yet thrown off its somewhat gruesome aspect. Useless, so-called palliative, operations on advanced cancer continue to be practiced, although the developments of radiation therapy render them less and less excusable. The frequency of deaths from salvarsan poisoning probably justifies the statement that this is the most dangerous remedy ever introduced into medicine. When we were in the army in Washington and were collecting diseased organs, we found this to be a frequent cause of death. I get reports from colleagues in various parts of the country who express the opinion that salvarsan collects a formidable toll of dead and injured. We have now to recognize the condition of delayed salvarsan poisoning, in which, two, three, or four months after the administration of salvarsan, the patient, apparently doing well, as far as syphilis is concerned, develops jaundice and pain in the liver, and dies of symptoms of acute yellow atrophy of the liver. Herxheimer has prepared an argument in defense of salvarsan, but these cases are of too frequent occurrence to make it probable that any other explanation is available.

[1] STRUMPEL. *Med. Klin.*, 1921, p. 1442.

There is no better illustration of the unwisdom of relying too much upon laboratory procedures to justify a serious therapeutic measure than in the matter of the transfusion of blood.

Blood transfusion was very popular in the army, but there we were dealing with healthy adults, and it was only necessary to choose the donors, do the typing, and take the necessary amount of blood. It was a most helpful procedure, was often life-saving, and very valuable. The hospital physician attempts to reproduce the same conditions in civil life with a different class of cases which are suffering from loss of blood from disease. He types the blood, gets the right donor and gives the right amount of blood in the proper manner, but the patient has a mild chill or a severe seizure, and in some instances the patient dies. They fail to recognize the other pathological conditions in the body, so that the blood of the donor is immediately hemolyzed and the results are disastrous. Therefore, greater care should be used in this regard; and the limitations of serological data should be recognized.

I am not at all competent to discuss the advantages and disadvantages of group medicine. There, again, I am quite content with the verdict given by those engaged in this branch of medicine after a reasonable trial. I encounter in my experience, however, some of the unfortunate results of this kind of medical practice. I find that it tends to increase the distrust of the public for the medical profession because it is very expensive to go through one of these groups and have everything done which can be done for diagnosis. It tends to develop laboratory investigations unduly and at the expense of direct clinical observation. I can cite the experience of a wealthy southern gentleman who was feeling not quite up to par and decided to come north and have an overhauling. He stopped at a well-known group clinic in a city about a hundred miles from New York. He spent one month there and everything that could be done in the way of laboratory procedures was done. At the end of that time it was found that nothing definitely wrong could be found with him, but he was advised to give up business and take a vacation. He then came to New York and submitted himself

to a group clinic here for three weeks (seven weeks in all) and his experience was exactly similar. He was informed that they too, were not quite certain of the nature of his trouble. They had several theories, but none of them was sufficiently tangible to warrant mention. They advised him, however, to quit work and stop smoking. The gentleman, feeling rather worse at the end of his experience said: "Doctor, I am very much disappointed, but I thought I would call your attention to a little swelling that I have noticed above my collar-bone that may be of some importance." Then for the first time he was subjected to an old-fashioned physical examination, first by the method called inspection, which revealed a swelling above the clavicle; then by another method known as palpation, which revealed a swelling, circumscribed in character, like a lymph node. This was followed by excision; then microscopic section of the mass by a pathologist, based on which followed a report reading: "Your patient is suffering from metastatic melanoma. Inspection will reveal a mole on the skin of the face or scalp. Some nodules will probably be found in other parts of the body. The prognosis is fatal. There is no treatment."

Most important is the unfortunate situation in hospital practice that results from the tendency of those in authority to do away with old-fashioned pathological services. I find that a very large proportion of the material resources of hospitals are now directed toward multiplication of laboratory methods of diagnosis, and to so-called clinical laboratory research. I have engaged in the pursuit of these methods myself and am responsible for one or two of them. They are now dead and buried or replaced by more modern methods. My pathological colleagues in all parts of the country agree that this phase of hospital practice has been seriously overdone. It is time to call a halt and evaluate the various methods which the given hospital is able to provide. We should maintain the function of the consulting pathologist and insist that every hospital that wishes to consider itself in Class A, as giving the most modern and safest service to its patients, must have a well-developed service in general pathology, whether it can afford other things or not. There are not many hospitals with such a service. As a rule, we

find that with the exception of a few of the larger hospitals in the larger cities, usually connected with universities, the men in charge of hospital laboratories are young men on very small stipends.

Now a man is not born with a knowledge of general pathology. He acquires it only after many years of laborious research and observation. It is a matter of experience. The greatest difficulty with hospital organization is that sufficient funds are not devoted to the development of competent pathologists. The positions where a man can get a living salary in the hospitals of today are very few. One hospital in New York has recently set aside a fund of $10,000 for the yearly salary of its pathologist. They can expect a good service in general pathology. Another well-known hospital, on the other hand, has decided to do without a pathologist. They farm out their difficult cases to different hospital pathologists in the city. They have no service in general pathology. They do not undertake to provide the salary of such a man. They are, however, very strong on pathological chemistry and chemical research. This is a very poor organization.

Now what are the functions of the hospital pathologist that render his services necessary? His first function should be to investigate the causes of the fatalities that occur in the hospital, to enumerate the lesions found, to elucidate the clinical course of the disease in connection with the lesions found, to correct partial or erroneous diagnoses, and to help to reduce the preventable deaths.

He should be especially concerned with the obscure cases that arise in the wards and operating rooms of the hospital. He should investigate them with all the means at the disposal of a well-organized pathological laboratory, including pathological anatomy, histology, bacteriology, chemistry and serology. He should keep himself familiar with the progress of the medical sciences and of clinical medicine in general, so that he may be able to furnish data from the literature. He should serve in many circumstances, especially if he has had considerable experience, as a general consultant in the wards and operating room. He should, by virtue of his knowledge of pathological anatomy, bacteriol-

ogy, serology and immunology, be able to furnish data with which, as a rule, his clinical colleague is less familiar.

He should engage in research. He should undertake to correlate the material at his institution and draw conclusions based upon series of cases and intensive study of single cases. He should contribute to the literature in this way and offer aid to his clinical colleagues to complete the study of their cases.

He should have entire charge of the clinical laboratory so as to be sure that the methods of clinical diagnosis are accurate and that the assistants are capable and reliable.

In conclusion I would venture to express the belief that:

1. Practitioners should be encouraged to rely more consistently on that great body of medical knowledge which is found in pathological anatomy and histology.

The tendency to displace this knowledge of the primary factors in disease by the pursuit of novel theories and venturesome therapeutics generally leads the practitioner astray. The interesting results of the great volume of laboratory research need not immediately be made the basis of practice. Some of these results may not stand the test of experience.

2. The hospital that attempts to do away with a reliable service in general pathology, and diverts its limited funds to the development of special and technical research, cannot deliver the best service to its patients. The multiplication of interesting but useless laboratory tests has reached such a point that most hospitals should carefully consider which of the many luxuries in laboratory investigation they can afford.

3. Medical schools should not subordinate the attested knowledge of pathological anatomy and histology, in order to meet the demands of other ambitious medical sciences. Medical students may be safely informed of the current results of much chemical, physiological and immunological research, only after they have become well grounded in the morphology of disease. Medical research suffers considerably from lack of attention to pathological anatomy and histology. The attempt to interpret disease from the exclusively chemical standpoint can never, in the nature of things, prove successful.

THE COMMON SKIN DISEASES*

JOHN A. FORDYCE, M.D.

THE following remarks accompanied a lantern-slide demonstration of some of the more common diseases of the skin, and are intended to emphasize certain features of interest to the general practitioner.

Skin diseases are largely neglected by the general practitioner, because they seldom endanger life. While this is true of many of them, others, like malignant growths, tuberculosis, pemphigus, exfoliative dermatitis and certain phases of a generalized syphilitic infection, do endanger life, impair health, or seriously handicap the patient's activities; but ordinarily skin diseases which come to the practitioner are usually free from danger. Another reason is that more acute and serious diseases absorb so much attention that little time is given to what are considered superficial and insignificant troubles. A third reason is the complicated nomenclature of skin diseases, which is so confusing that medical students and practitioners make little or no effort to master it.

The etiology and treatment of the more usual skin diseases may easily be comprehended by attention to a few general principles without committing to memory the technicalities of this specialty.

Many erroneous views prevail in the profession, as well as among the lay public, as to the cause of skin diseases. Dietetic errors and excessive uric acid in the blood are too often incriminated as factors without more reason than tradition and impression. Our modern conception of protein sensitization corroborates to a certain degree the older views as to the relationship of diet to certain outbreaks on the skin. Urticaria and serum rashes are typical examples of this type of reaction.

*This lecture was delivered on Apr. 28, 1922.

Acne

Acne is one of the common skin diseases, and one about which erroneous impressions prevail. Parents often believe that acne is a disease of a certain age and connected with repression of the sexual function. Furthermore, they are told by physicians that it is an insignificant trouble which usually cures itself. It does cure itself in many cases, but often with disfiguring scars, as will be shown in the

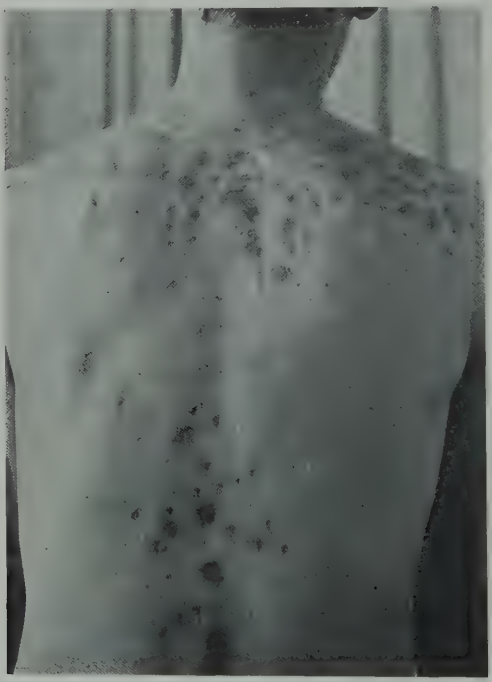


Fig. 21. Extensive scarring resulting from a severe case of acne indurata.

accompanying illustrations (Figs. 21 and 22). This aspect is often lost sight of, and many a girl or boy is marred for life by neglect of proper treatment. Scars which follow cure of active pustular lesions are not infrequently attributed by the patient to the procedures employed by the physician. Before treatment is begun this possibility should be clearly stated to the patient. Scars result from destruction of tissue in and about the pilosebaceous follicles, and not from the thera-

peutic methods. The factors which cause acne, a disease of age incidence, are: the active development of the hair follicles and sebaceous glands at puberty, plus an infection of these structures with the acne bacillus and ordinary pus organisms. If acne is looked upon as a local infectious process, it can be cured. If it is considered to be a constitutional disease, and treated as such, it is seldom cured, or only after destruction of the involved glands. Little progress, therefore, can be made in treating acne by attention to diet, the state of

FIG. 22. Acne vulgaris showing scarring of the face and active lesions on the neck.

the bowels, or remedies directed to a theoretical blood condition. It must be treated as a local infectious process.

In treating it the prejudice against the use of soap and water on the face must be overcome. Soap is one of the best remedies. It opens the orifices of the sebaceous glands and enables the remedies to penetrate more readily. Furthermore, acne of the face, back and chest is not infrequently complicated with a dry or oily seborrhea of the scalp. If our therapeutic endeavors are directed only against the eruption on

the skin, we may have relapses because re-infection from the scalp takes place. The scalp, therefore, should be treated at the same time.

The widespread prejudice as to the injurious effect of washing the scalp has no logical basis. The hair on the scalp is not injured by washing any more than the face or body hairs.

In treating acne, always keep in mind that you are dealing with a local infection, and one which is inoculable from one part of the skin to another. It is for this reason that the disease is frequently spread in so-called beauty parlors by the foolish practice of skin massage. By this procedure the organisms are spread from one part of the face to another. Superficial pustules and deeper abscesses should be carefully opened and the contents expressed, after preliminary washing of the face by soap and water. After the abscesses have been opened and disinfected, and the comedones expressed by a suitable instrument devised for the purpose, the face should be thoroughly disinfected by the use of the well-known lotio alba. I usually employ this preparation in much stronger concentration than given in the textbooks. A saturated solution may be made by first dissolving in water equal quantities of each ingredient, namely, sulphate of zinc and sulphuret of potash, and then mixing the two solutions. This forms a very dense precipitate which is applied to the skin. The patient is furthermore directed to use this solution once a day and to alternate it with an ointment containing 40 grains of precipitated sulphur and 20 grains of salicylic acid, to an ounce of some ointment base. After these remedies and procedures are used for ten days or two weeks, a marked improvement will be noted in the condition of the skin.[1] At this time it is my custom to employ fractional doses of *x*-rays. I use one-quarter of a skin unit, carefully measured by our modern apparatus, at weekly intervals, after the method worked out by Dr. George M. MacKee. From eight to twelve exposures of these small doses of *x*-rays complete the cure. Relapses, however, occur from time to time and should

[1] Further experience seems to show that the preliminary use of sulphur ointment renders the skin more liable to pigmentation. Although this is not permanent, it is perhaps advisable to omit this application before and during *x*-ray treatment.

be treated in the same manner. Or they may be controlled by the Alpine lamp, thus obviating additional *x*-ray therapy. By persisting in treatment along these lines, the disfiguring scars can be prevented and a practical cure of the condition brought about.

In the early days, when *x*-rays were used regardless of the amount, much harm was done to the skin by burns of an acute character, or by slower degenerative changes that led to telangiectasia and atrophy of the skin—conditions which are irremediable.

Coincident seborrhea of the scalp should be treated by applications of the sulphur and salicylic acid ointment twice a week, or by lotions containing resorcin with bichloride of mercury.

Another disease of the skin, frequently referred to as acne rosacea, comes on at a later period of life, and sometimes leads to disfiguring conditions of the nose, known as hypertrophic rosacea or rhinophyma. This can be cured by surgical measures. The milder types are curable by attention to diet and the local use of astringent or healing applications. Multiple scarification is often useful.

Impetigo

Impetigo is a term used to indicate infection of the skin by the various types of pus organisms.

The so-called follicular impetigo of Bockhart is caused by infection of the hair follicles with ordinary staphylococci (Fig. 23). The more common type seen about the face, but which may involve any part of the cutaneous structure, is known as contagious impetigo. It is not infrequently mistaken for eczema. It begins as a vesicular or bullous lesion containing clear fluid which soon becomes turbid, and dries in the form of superficial crusts. It is contagious as well as autoinoculable. It is carried to various parts of the body by the hands, toilet articles or infected clothing.

Some cases of impetigo contagiosa simulate ringworm because the epidermis is undermined by the organism. The center of the lesion heals, but the periphery shows an active inflammatory reaction (Fig. 24). It is acquired at times in the barber shop and is called by the laity "barber's itch." It is,

however, not a fungus infection, but one primarily due to a streptococcus of rather low virulence. To this infection is superadded one with the ordinary skin staphylococci, so

FIG. 23. Staphylococcic impetigo. (Bockhardt type.)

that when the crusts are formed, we have a combination of the two infections.

In the newborn a generalized exfoliative dermatitis is sometimes met with—the so-called Ritter's disease—which

is probably due to the same etiological factor as impetigo contagiosa, or to staphylococci alone; but on account of the delicate structure of the infant's skin a reaction of a different type takes place. When the organisms in Ritter's disease invade the blood stream a generalized infection takes place leading to the death of the infant.

Other varieties of impetigo are pemphigus neonatorum, in which the eruption is a generalized bullous one occurring in newborn children and simulating pemphigus, and ecthyma, usually seen on the extremities, a deeper and more severe

Fig. 24. Impetigo contagiosa (circinate type).

form of impetigo, which often causes destruction of a portion of the corium.

Impetigo contagiosa, as usually seen about the face, is one of the easiest of skin diseases to cure, and one for which we have a specific remedy, namely, ammoniated mercury ointment. If this is applied to the infected surfaces after the crusts have been removed with oil or boric acid compresses, healing takes place within a few days. In extensive cases of impetigo which are occasionally met with

over a large extent of the body surface (Figs. 25 and 26) ammoniated mercury is not a safe remedy to apply. I have found that in such cases the application of dilute tincture of iodine or of iocamfen (a combination of iodine, camphor and phenol) acts rapidly and in a satisfactory manner.

A number of lantern slides were shown to illustrate types of catarrhal dermatitis, which included dermatitis due to various external irritants.

FIG. 25. Bullous impetigo contagiosa.

The number of plants that produce dermatitis of the skin is very large. The ones best known are poison ivy and poison sumac. At certain seasons of the year the florists' shops display the primrose in great abundance. In susceptible individuals a dermatitis, very similar to the type produced by ivy and sumac, may follow exposure to this plant. Many cases of plant poisoning have been wrongly diagnosed by physicians and called eczema. These patients have been treated on general principles under the supposition that some

poison in the blood was responsible for the outbreak on the skin. A careful examination of the patient's surroundings often clears up the etiological mystery. A dermatitis on the

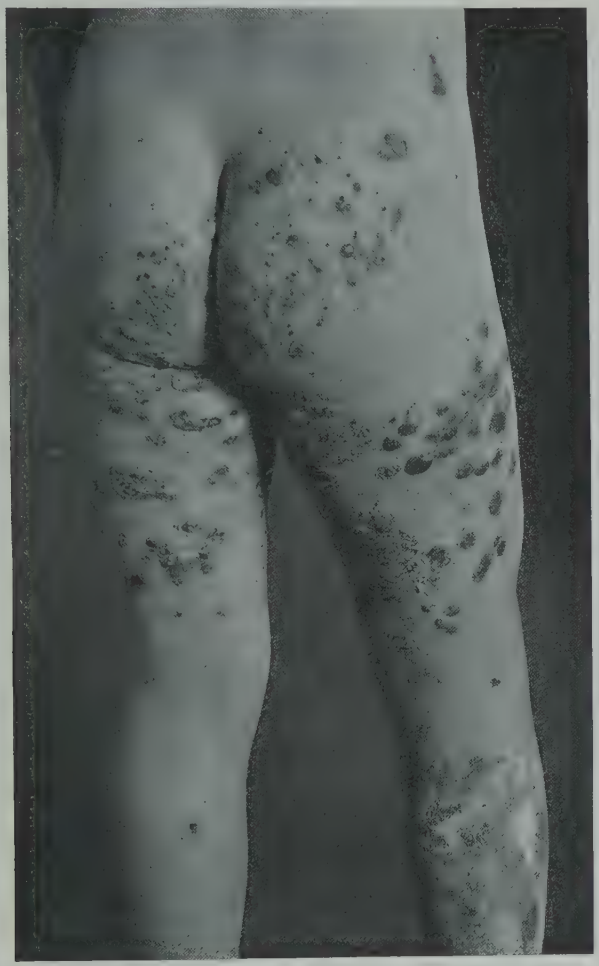


FIG. 26. Bullous impetigo contagiosa.

hands and face occurring in the late winter and early spring should lead one to inquire if the patient has a primrose in the house. It is surprising to note how often one will get a reply in the affirmative.

In the treatment of dermatitis due to exposure to poisonous plants, the best procedure is to cleanse the surface of the skin thoroughly with soap and water, then apply 95 per cent alcohol to neutralize the poison; following this by compresses saturated with calamine lotion, plus the addition of a certain quantity of a dilute solution of subacetate of lead.

The term eczema means very little. It is largely employed by the general practitioner to cover his ignorance of possible

FIGS. 27 AND 28. Eczematoid ringworm (epidermophytosis).

external factors. Most of the cases, if investigated, will be found to depend upon some external cause. Some types of eczema met with in old people may be due to a constitutional defect, but of such constitutional conditions we know very little.

There is a type of dermatitis seen on the hands and feet which is attended with the formation of deep-seated vesicles, and later, by larger patches of inflamed skin, which may assume either a moist or a dry character. This type of catar-

rhal dermatitis is sometimes met with in connection with eczema marginatum of the inguinal regions or axillary spaces. The association of the two conditions led Sabouraud, a French dermatolgist, to investigate the hand and foot lesions. He found in many of them a fungus identical with the fungus causing eczema marginatum. This eruption, known as eczematoid ringworm, often begins with lesions between the fourth and fifth, or the third and fourth toes. The

FIG. 28 *A*. Eczematoid ringworm (epidermophytosis).

epidermis there is thickened and sodden and often fissured. From this focus of infection the dermatitis spreads to the soles and dorsal surfaces of the feet (Figs. 27 and 28) and from the lesions between the fingers to the hands and forearms (Fig. 28 *A*). It includes many cases formerly called pompholyx, often noted as a relapsing affection over many years. Secondary vesicular and scaling lesions in connection with those of the feet and hands are at times met with over a large part of the cutaneous surface and may be due to some irritant produced by the fungus or to a possible infection through the blood. It is difficult or impossible to demonstrate fungi in the secondary lesions (Fig. 29). Whether all cases of vesicular disease between the fingers and on the feet are due to a ringworm fungus has not been definitely decided. It is an extremely difficult affection to cure, because

once the fungus is implanted in the skin it is not easy to bring our remedies in direct contact with it. By persisting in the use of local remedies, which are those usually employed in ringworm infection, and supplementing this treatment with the use of *x*-rays in the proper dosage and at stated intervals, many of these cases may be cured.

In referring to these types of dermatitis where an external irritant has been definitely proved, I am endeavoring to emphasize the importance of searching for a local cause

FIG. 29. Secondary vesicular and scaling lesions in eczematoid ringworm.

when a case of catarrhal dermatitis, which we call eczema, presents itself to the practitioner.

Certain individuals handle heavy paraffin oils and their clothing becomes oil-soaked. This oil-soaked clothing in contact with the skin produces various types of dermatitis.

When a patient comes to you with a dermatitis about the forehead and eyelids, which spreads to the face, and when the color of the hair does not look natural, or where the hair is dyed, suspect the effect of a hair dye.

One of the most commonly employed dyes at present is an anilin preparation sold under various trade names, which

dyes the hair a titian red and various shades to black. It produces in susceptible individuals not only a dermatitis, but also general symptoms, such as headache, vomiting, vertigo, blurring of vision and palpitation of the heart. Several years ago I saw a patient, with a well-known practitioner in New York City, who used this preparation on the hair and who developed, in addition to the dermatitis, the symptoms which have been mentioned. She had been sent to several health resorts to recover her nervous equilibrium. The entire picture was caused by the para-phenalindiamin solution which had been used for dyeing her hair.

The hands of barbers who rub certain hair dyes and tonics into the heads of their customers are sometimes affected with a vesicular or scaling dermatitis.

Individual susceptibility is one of the factors which influences the development of dermatitis of this type, just as in ivy or primrose poisoning.

Nurses in hospitals, or laboratory workers, who come in contact with formaldehyde, may develop a dermatitis which continues as long as they pursue their occupation. They become so sensitized to this chemical that the smallest quantity causes a skin reaction.

In carpenters and joiners the resinous dust of certain hard woods, like teak, ebony, satin wood and rosewood, will occasionally set up a severe dermatitis.

The chief occupational diseases of the skin are known as trade eczemas and are due to the direct irritant action of chemical, thermal and physical agents. While frequently of little significance they sometimes are so serious as to compel a change of work.

Certain individuals exhibit a high degree of sensitiveness which becomes heightened by continued exposure. Bakers, varnishers, bricklayers, masons (Fig. 30), sugar workers, metal workers, printers, machinists and workers in various chemicals develop these catarrhal skin inflammations. In furriers, sulphide of arsenic used in curing furs sometimes produces a severe inflammation of the skin with persistent ulceration. Dyers and workers in anilin colors are apt to develop eczematoid and pustular eruptions. Black, red and orange pigments are particularly irritating, and articles of apparel which

come in contact with the skin may cause an inflammation difficult to interpret unless this possibility is borne in mind.

Tar and paraffin workers develop on the hands and arms rough thickened patches on the skin which may eventually lead to warty growths and cancerous degeneration.

A series of slides were shown at this point, illustrating another type of dermatitis, formerly included in the eczema group. This particular form of eczema, which I call infectious

Fig. 30. Dermatitis venenata of hands from lime.

eczematoid dermatitis, is seen after abscesses, discharging sinuses following operations, or from any focus in which pus is present. It spreads by autoinoculation and produces groups of vesicles that run together and cause large eroded surfaces discharging a clear serum or seropus. It is probably due to staphylotoxins. Infection with the staphylococcus alone leads to pustular affections. The toxins associated with the organism produce a catarrhal disease of the skin simulating very closely what we commonly know as eczema. This condition is seen very frequently in hospital patients or in patients who come to the hospital from lodging houses and who pay little attention to the personal hygiene of the

skin. Their clothing is frequently soaked with pus from leg ulcers or from abscesses which follow pediculosis of the body (Fig. 31). The condition yields fairly rapidly, after thorough cleansing, to an ointment containing about 20 grains of sulphur to the ounce of Lassar's paste.

It is important, therefore, from an etiological standpoint, to separate the various types of catarrhal dermatitis as far as possible. It is only in this way that success in treatment can be achieved.

FIG. 31. Eczematoid dermatitis in a lodging-house patient from clothing saturated with pus.

After scabies, or in association with it, we very frequently see eczema about the breasts of women. (Fig. 32). The scratching of the skin and abrasions of the epidermis lead to infection with pus organisms.

In infantile eczema a number of types are met with. In some cases it apparently starts from scalps which are not properly looked after and where the vernix caseosa is not removed. Organisms are harbored beneath the crusts, and from this focus we see moist lesions and dry scaling patches

spread over the neck, face, arms and trunk. These cases can be cured by attention to the point of origin. A small amount of ammoniated mercury in a boric acid or zinc ointment often succeeds in controlling the outbreaks.

Where eczema in infants appears on the cheeks and from there spreads over the face and other parts of the body, overfeeding with sugar or fats or a sensitization to some food product, may be the etiological factor. Examination of the stools, to determine whether certain food constituents are

FIG. 32. Eczematoid dermatitis of the breasts secondary to scabies.

badly digested, may throw some light on these cases. I have never, however, been able to derive much benefit from investigations of this kind. I have been in consultation with many good pediatricians, and have seen the food formula changed many times, the stools analyzed and sensitization tests made, but the application of the results made by these various investigations has not been of much help to the patient.

The best remedy for infantile eczema of this type with which I am acquainted is a remedy known as naftalan,

a petroleum preparation originally of Russian origin. It has the consistence of vaseline and somewhat resembles ichthyol. Naftalan may be used as an ointment base to which zinc oxide and starch may be added. If to this formula we add a small amount of coal tar, we have a good preparation for this form of infantile eczema.

The proper handling of infantile eczema requires a great deal of ingenuity in the use of remedies, and is oftentimes followed by failure to obtain results. Some of these patients undoubtedly are sensitized to some protein and may continue to have relapses during many years. Radiotherapy at times influences these eruptions with astonishing rapidity.

There is another type of dermatitis, known as seborrheic eczema, that is associated with certain pathological conditions of the scalp, the most common of which is the so-called dry dandruff. From the scalp as a center, the affection spreads to the forehead, back of the ears, and to the back and front of the chest. On the scalp it sometimes assumes a moist form, but on the body it usually produces patches sharply circumscribed and covered with scales more or less greasy. This type of dermatitis yields readily to a combination of sulphur and salicylic acid in ointment form. In association with the scales on the scalp, various types of organisms are found, some of which are probably responsible for the persistence of the trouble there and for its spread to the parts which have been mentioned.

Several slides were here shown illustrating a condition of the skin called lichenification, a leathery-like condition seen at the margin of the hair, on the neck, the bends of the elbows (Fig. 33) and on various parts of the body. It may develop as a primary condition preceded by marked pruritus, or as a secondary one after various types of dermatitis. The patient scratches incessantly and the scratching is followed by thickening and pigmentation of the skin. This condition is difficult to cure by the use of topical remedies, but may be controlled by the proper use of *x*-rays or the procedure I will shortly mention.

A tradition prevails that soap and water should never be employed in eczema. This, I believe, originated from a statement of Erasmus Wilson forty or fifty years ago. He

meant, I think, that soap and water should not be used in acute catarrhal dermatitis where the horny layer is absent, where the rete is exposed and where soap and water soften the epidermis still more and produce irritation. Because of this statement by Erasmus Wilson, many practitioners prohibit the use of soap and water in any skin disease without logical reason.

In cases of chronic eczema where vesiculation no longer exists, or where it is concealed beneath a thickened epidermis,

Fig. 33. Lichenification of the skin.

the use of soap and water is one of the best preliminary remedies we can utilize.

In lichenification of the skin, thorough rubbing with tincture of green soap is a good introduction to the use of ointments containing tar or other stimulating remedies.

I have cured patches of chronic lichenified skin by applying to the surface a 15 or 20 per cent solution of caustic potash. This is rubbed on the skin and the softened epidermis curetted away. Following this preliminary treatment with caustic potash and the curette, a 50 per cent ichthyol solution

painted on the surface and permitted to dry by the addition of a powder or absorbent cotton, forms a dressing which remains in contact with the surface for several days. A repetition of this procedure is often successful in curing patches of chronic thickened skin.

In the last few years *x*-rays have come more and more into therapeutic use in a large majority of skin diseases. The results are often rapid and astonishing, not only to the physician, but to the patient as well. In the very superficial forms of dermatitis, such as eczema, we employ a quarter of a skin unit given at weekly intervals. Four to six treatments may be sufficient. This rule applies not only to the moist catarrhal types of dermatitis, but to the dry forms seen in psoriasis. In deeper-seated affections which extend below the epidermis, half a skin unit may be employed and repeated at intervals of two weeks. In the treatment of new growths, two or more units are employed and the intervals correspondingly lengthened.

The use of *x*-rays has revolutionized the treatment of ringworm of the scalp. It is here given in doses of one unit—the so-called erythema dose. The hair falls out in from two to three weeks, and, as a rule, the ringworm is cured. This method of treatment was devised by Sabouraud of Paris. It can be employed only in children who have sufficient self-control to keep quiet during the application. If too large a dose is used, as happens if one is not experienced in its application, a permanent alopecia will be produced, but where it is properly used, the hair returns in about three months.

Careful measuring of the doses of *x*-ray, as well as regulation of the intervals between applications, enables us to accomplish vastly more than we formerly did, and with none of the unfortunate results that followed the haphazard use of this agent.

Slides illustrating the various types of ringworm infection of the skin were shown, including eczema marginatum of the axillary region with secondary or satellite lesions scattered about the primary growth (Fig. 34). This illustration was an excellent example of autoinoculation of the skin. A similar condition is not infrequently seen in psoriasis and suggests in this trouble a local factor as the causative agent.

FIG. 34. Eczema marginatum of the axilla (epidermophytosis.)

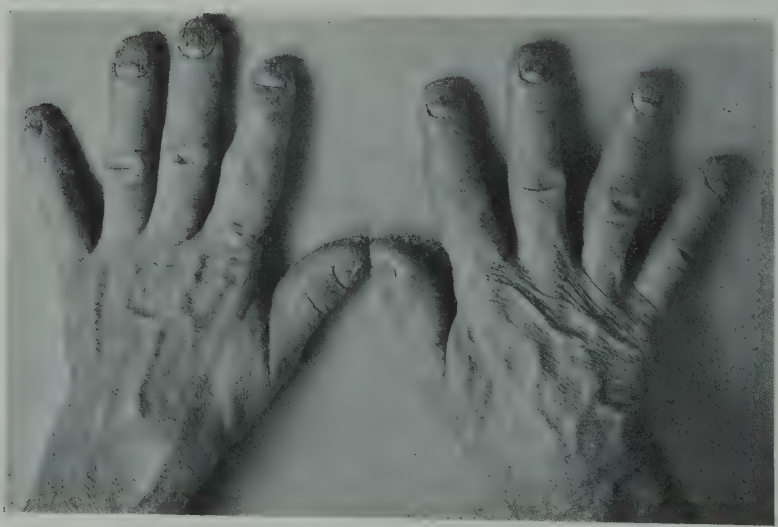


FIG. 35. Ringworm of the nails.

Several illustrations were shown of the various types of ringworm of the scalp, including the small spored ringworm which leads to broken and lusterless hairs on round or oval scaling patches in the scalp. The ringworm fungus has a

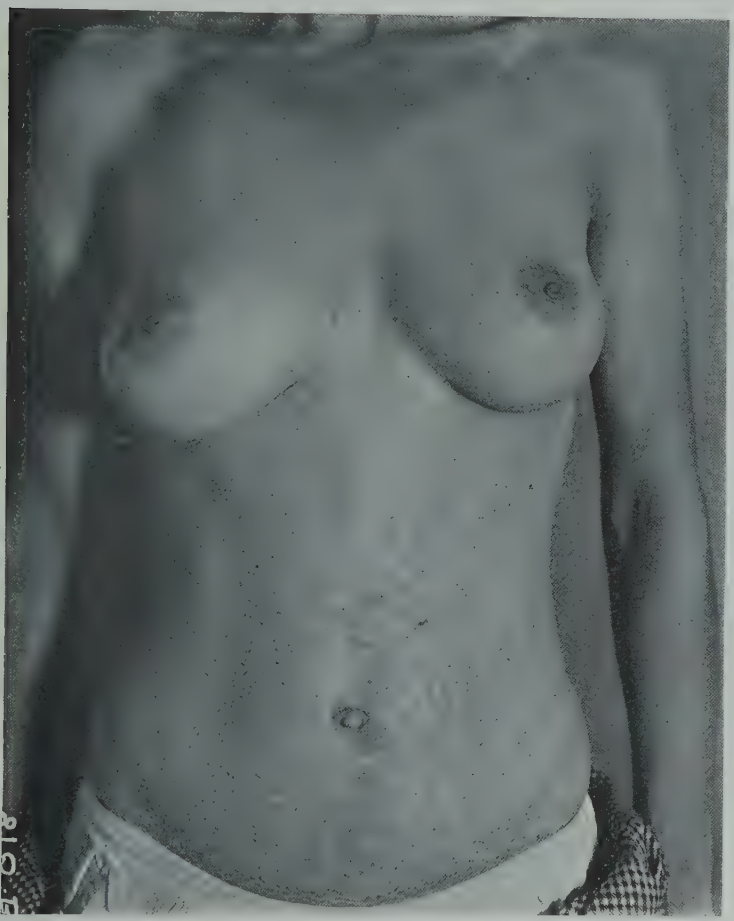


Fig. 36. An unusual form of pityriasis versicolor involving the follicular orifices.

special affinity for the horny layer of the skin. It involves the nails and leads to a condition sometimes difficult to diagnose and more difficult to cure (Fig. 35). It is at times necessary to remove the nails in order to eliminate the fungus. In the

beard the fungus penetrates the stiff hairs and produces elevated boggy swellings which simulate abscesses.

Another fungus infection of the skin, tinea versicolor, was illustrated in the lantern demonstration. In this affection no inflammatory reaction occurs in the skin. A discoloration is met with and a branny scaliness can be demonstrated by scratching the surface (Fig. 36). These scales, if examined in a properly prepared preparation, show the typical fungus. The affection in question is usually readily cured by scrubbing the surface with soap and water and afterward using a satu-

Fig. 37. Erythema multiforme (typical site).

rated solution of hyposulphite of soda. Recurrences are not infrequently met with if all the lesions are not cured. It is very difficult to exterminate the last group of spores, and these form a focus for a relapse or an extension of the condition. This principle applies to many skin diseases.

Another type of skin eruption, known as erythema multiforme, was illustrated in the lantern slides. This condition occurs by preference on the hands and feet (Fig. 37). It produces red elevated lesions, some of which may become bullous or hemorrhagic. It simulates very closely an urticaria,

but the individual lesions persist a much longer time. It relapses from time to time and originates probably from some focus in the body, such as a pathological condition of the tonsils, or pus in a confined location. It is at times, however, difficult to determine an extra-cutaneous focus. One type of this eruption occurs not only on the skin, but involves the lips and the mucous membrane of the mouth. This variant is known as erythema iris. It is occasionally attended with constitutional disturbances, such as elevation of temperature. I have seen in some of these cases a temperature of 105° F. persist for several days. Deaths have followed with negative autopsy findings.

Erythema multiforme is seen in the course of acute rheumatism, and here we have a definite etiological factor to deal with. The great majority of cases, however, which come to the dermatologist, give no history, and have no evidence, of a rheumatic affection.

In erythema nodosum, which is accompanied by joint manifestations, the involvement of the joints may be of a similar nature to the lesions on the skin, and not of rheumatic origin.

In certain types of cardiac disease of streptococcic origin, invasion of the blood-stream by the hemolytic streptococcus leads to types of purpura. A remarkable example of this came under my observation some years ago, in which not only was the skin involved, but hemorrhagic lesions were present beneath the conjunctiva and in the mucous membrane of the mouth and nose.

Scabies

I have recently had under my observation several girls from various schools about New York who had typical scabies. One girl, while on her vacation, developed an itching eruption on the skin. Her physician told her it was due to uric acid. She returned to her school and eighteen other girls developed scabies. This experience is not unique; hence I am trying to impress upon my students the importance of recognizing the more common diseases of the skin, and especially those which endanger the surroundings of the patient.

Years ago I practiced medicine in a country town and was associated with an old doctor who had been trained in dermatology. He was accustomed, at times, to the excessive use of alcohol, and when under its influence told me the story about a case of itch that came to him. He sent this patient to every doctor in the town, but all of them failed to make the proper diagnosis. The old doctor used to take a great deal of pleasure in repeating this story, adding that

Fig. 38. Typical scabies lesions.

if the physicians in the town could not diagnose itch, they were not very safe advisers in more serious troubles.

At times we fail to find the typical lesions of scabies between the fingers or on the wrists. It is not always possible, either, to demonstrate the characteristic burrow. We make a diagnosis by the distribution of the eruption and by the intense itching, always aggravated at night. Furthermore, if more than one case of a persistent itching eruption with a rather wide distribution is found in a family, it is safe to assume that we have to do with scabies.

The lantern slides that were shown illustrated some of the more serious and also some of the milder forms of the trouble (Fig. 38).

When the affection is neglected, we not infrequently have secondary infections consisting of pustules and patches of eczematoid dermatitis (Fig. 32).

Pityriasis rosea is a rather common skin affection which has a distribution over the trunk and on the extremities as far as the elbows or knees (Figs. 39 and 40). It very closely simulates ringworm, and was for a long time regarded as a

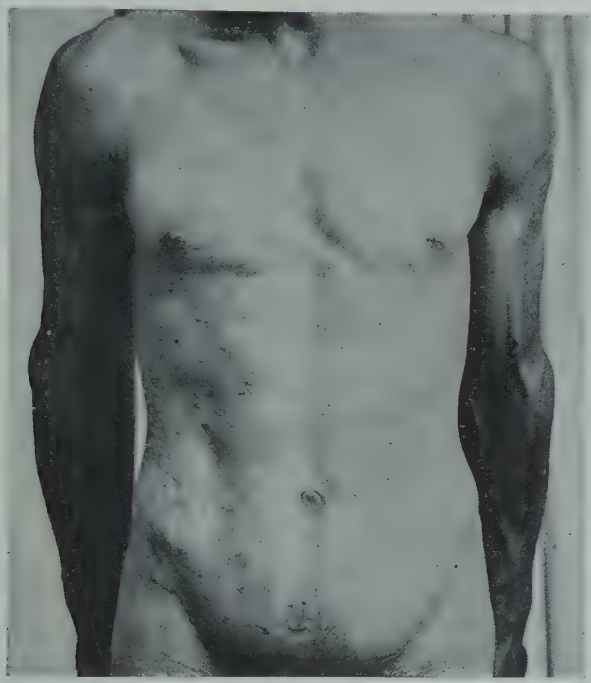


FIG. 39. The circinate type of pityriasis rosea.

disseminated form of this affection. It develops as a circinate or macular type. It has also been mistaken for an early syphilitic eruption. No organism has been found in the scales, but its mode of origin from the so-called herald or primary spot suggests a local infection. It is important to recognize it and to employ the correct treatment, because if it is called ringworm and the ordinary remedies used in ringworm are employed, intense irritation of the skin may follow. In other words, it eczematizes easily under the

influence of sulphur or other irritants. The best treatment to employ is some mild application like calamine lotion. The duration of the trouble is from four to six weeks, or longer. No constitutional disturbance attends it.

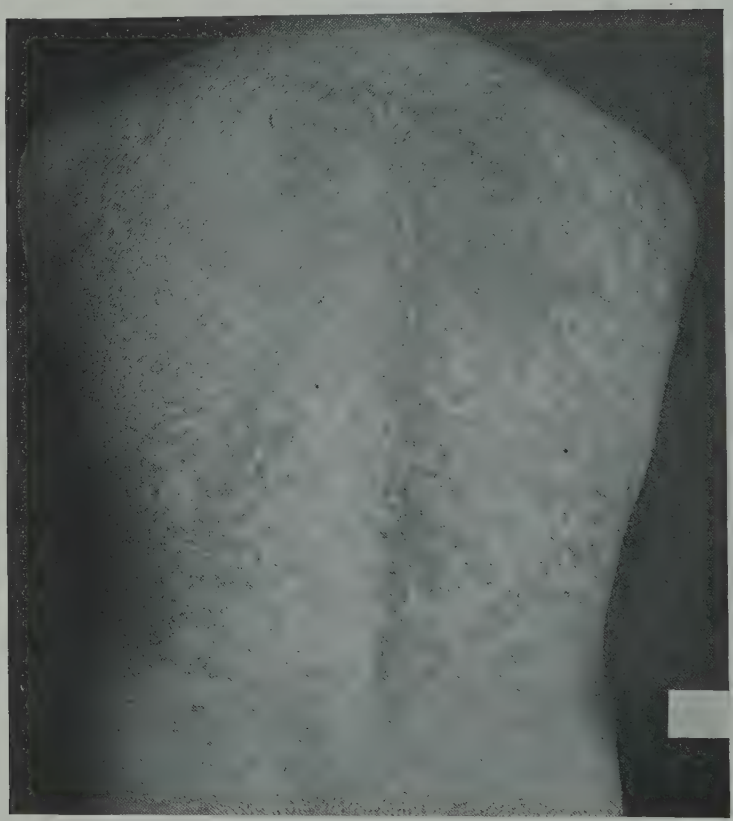


Fig. 40. Pityriasis rosea; macular type showing confluence of lesions on the upper back.

One of the most distressing of the common skin diseases is psoriasis, which assumes a large number of clinical forms (Fig. 41). It frequently begins at an early age and relapses, often during the lifetime of the individual. The existing eruption can be removed by the use of remedies like chrysarobin, pyrogallol or the tars. On the scalp the best remedy is a 25–50 per cent ointment of ammoniated mercury with

or without the addition of salicylic acid. This must be used with a certain amount of circumspection and observation, because absorption may take place and lead to a mercurial

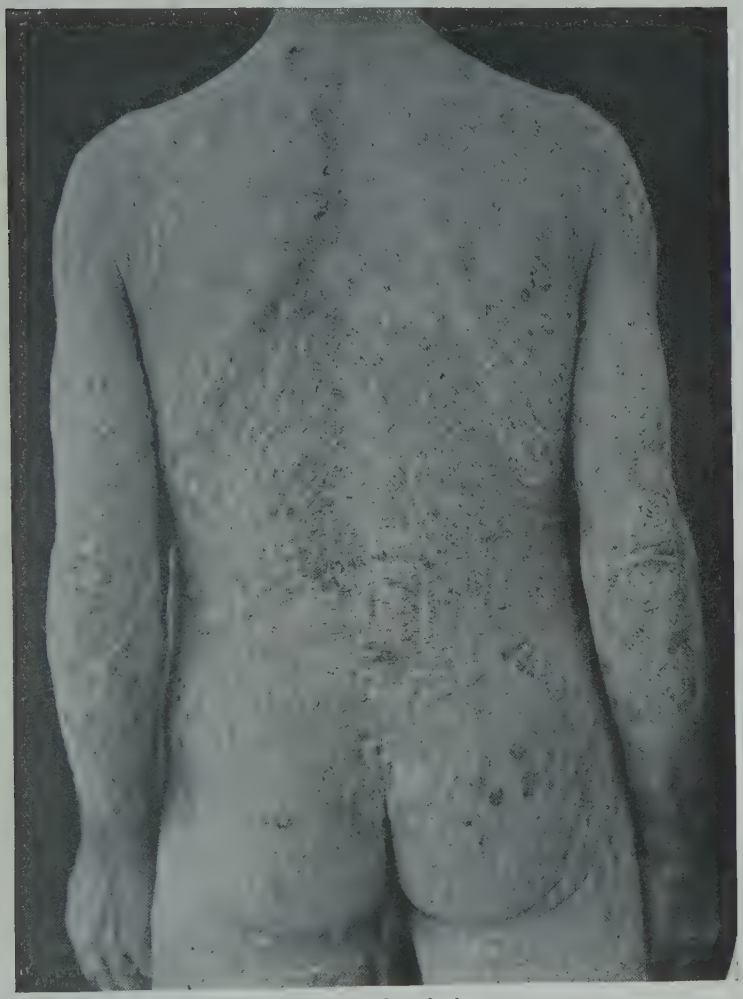


FIG. 41. Psoriasis.

stomatitis. *X*-rays in fractional doses rapidly clear up the lesions, in many cases, but relapses occur just as frequently after *x*-ray treatment as they do after the various topical

FIG. 42. An extensive bromoderma in an epileptic to whom the drug had been administered over a long period.

applications which have been mentioned. Some cases are resistant to *x*-rays, and this treatment should not be persisted in if results are not soon apparent. If care is taken, however, to clear up all the lesions on the body, the intervals between relapses grow longer.

Eruptions from the ingestion of various drugs are so numerous and multiform that it is impossible in my time

FIG. 43. Persistent erythematous lesions from the use of cathartics containing phenophthalein.

limit to discuss them. The illustration, Figure 42, is a remarkable example of a widespread pustular and fungating eruption from the long use of bromide of potassium. Eruptions after the administration of the bromides and iodides are more constant than after any other medicaments, and may

simulate lesions of a different etiology. The iodides may cause ulcerative and encrusted lesions like a rupial syphilide, or deep-seated subcutaneous swellings very similar to gummata. A knowledge of the possible effects of drugs on the cutaneous surface often enables us to interpret a puzzling skin problem.

In 1916, several patients presented themselves at the Vanderbilt Clinic with patches of marginate erythema over the trunk and extremities. This partially disappeared, leaving a rather persistent pigmentation, but it recurred in the same sites. These cases were presented by Dr. George M. MacKee and Dr. Fred Wise, before the New York Dermatological Society and the New York Academy of Medicine, under the titles of "Erythema Multiforme" and "Erythema Multiforme Perstans." In searching for a possible etiological factor it was found that these patients had taken cathartics containing phenolphthalein. It was also found, by administering phenolphthalein to patients in whom this type of eruption occurred, that the patches again appeared. Dr. E. W. Abramowitz, who is connected with the Clinic, reported the cases under the title of "Erythema Multiforme Associated with Cutaneous Pigmentation" and recognized the etiological connection of the drug with the eruption in question. These phenolphthalein eruptions were subsequently discussed in an article by Dr. Wise and Dr. Abramowitz in the *Archives of Dermatology and Syphilis*, March, 1922. The eruption in question presents many points of similarity to the one which occasionally follows the ingestion of antipyrine.

I realize that this formal talk is fragmentary and incomplete, and perhaps open to criticism. It may serve, however, to correct some erroneous views as to the common diseases of the skin, and emphasize the importance of eliminating a local origin before incriminating general factors, which are too often vague and indeterminate.

DIAGNOSIS OF ABDOMINAL TUMORS*

RUSSELL S. FOWLER, M.D.

UNTIL the happy day arrives when people with trifling deviations from normal physiology will consult physicians thoroughly qualified to make examinations, physicians as now qualified will be obliged to make, in a large majority of their cases, diagnoses based upon end-results of what was indicated in its incipiency by a slight error in physiology.

The medical profession and the public will require many decades of education before the happy period is reached in which the thoroughly skilled physician will painstakingly examine patients in whom only the microscope, the roentgen ray, the illuminating instrument, or the physiological laboratory can determine disease in its incipience.

The diagnosis of the obvious is the diagnosis of today; the diagnosis of the future will be the diagnosis of underlying errors in physiology. Until that happy millenium, we must deal with the obvious.

Abdominal tumors are obvious and in this lecture we will deal with the most obvious of them.

GENERAL CONSIDERATIONS

As Osler says, "In the diagnosis of abdominal tumors Bishop Butler's maxim that 'probability is the rule of life,' is particularly true, and the cock-sureness of the physician who formerly had to dread the mortifying disclosures of the post-mortem table is now wisely tempered by the surgeon who can so safely and promptly decide upon the nature of an obscure case."

* This lecture was delivered on May 18, 1923. Over one hundred specially prepared slides were used in illustrating the lecture. It is impossible to reproduce them all in connection with the printed lecture. Many of them were made to show the appearance of the patient presenting a tumor with its surface characteristics, following which a slide was presented showing the tumor in situ in natural colors and relationship seen through a transparent abdominal wall.

The surgeon of today has a most decided advantage over the surgeon of former days. He is becoming more and more of a diagnostician, whereas the surgeon of years ago was but the servant of the physician. The surgeon of today has learned to correlate physiology, pathology and symptomatology in the living. I can conceive of no greater education in diagnosis for the family physician than a course yearly in a surgical clinic. Yet fewer and fewer physicians attend operations upon their patients. I can remember that twenty-five years ago it was the universal custom for each family physician to attend the operation on his patient. As far back as ten years ago the custom was still fairly well followed. But since that time less and less adherence to that custom has been observed. As for attendance at operations in general, the family practitioner is noted for his indifference.

For this reason the didactic lecture of former years is being replaced by clinical lectures with demonstration of patients, or apparatus, specimens or pictures which may to some extent take the place of the better facilities afforded at the operating and post-mortem tables.

In entering upon a study of abdominal tumors it is fundamental that the anatomy of the abdominal cavity must be known; for the first consideration in the diagnosis of abdominal tumors, after establishing the fact that a tumor is present, is the determination of the organ involved. The normal location and characteristics of each of the abdominal viscera must be known. Many tumors correspond both in position and shape to the normal organ (tumors of the gall-bladder, liver, spleen and kidney).

Certain tumors can be identified with the organ to which they belong, by their resemblance to the normal organ in outline, edge and consistency, even though they do not occupy the normal site of that organ.

On the other hand, a displaced viscus may be mistaken for a tumor, as for instance, a movable kidney in the pelvis or a floating spleen in the pelvis.

Occasionally a distended bladder is mistaken for a tumor.

The *probability* is that the organ involved is the one which normally occupies the area in which the tumor is found.

The second consideration is the nature of the growth.

To answer these questions we must accumulate certain data. First, the history of the case; second, the examination of the abdomen; third, the results of special examinations (inflation of the stomach and colon, cystoscopy, radiographic examination, laboratory tests) made in accordance with the organ or organs suspected.

HISTORY. Age, habits, venereal history, prior illnesses, previous operations, variations in weight, enlargement of the abdomen and the rate of enlargement, pain and other symptoms relating to deviations from normal physiology.

EXAMINATION OF THE ABDOMEN. As in diagnosis in other regions, the diagnosis of tumors of the abdomen rests upon inspection, palpation, percussion and auscultation.

Rectal examination should be made in practically all cases. It is of value not only in aiding to locate the tumor, but also in determining whether or not there are secondary carcinoma deposits in the pelvic glands. In children, rectal examination is of special value on account of the small size of the abdomen. A great deal of information can be obtained by the examining finger. Vaginal examination is of aid in differentiating tumors in the pelvis from tumors in the abdomen proper, and in demonstrating the existence of other lesions. Cystoscopic examination is a necessity in cases in which tumor of the urinary organs is suspected. In doubtful cases, inflation of the stomach and colon will aid.

There are certain conditions which render examination of the abdomen difficult. *First;* rigidity of the abdominal wall. This may be caused by struggling on the part of the patient, as in infants or young children. It may be the timidity in young nulliparous females, or the well-toned muscular rigidity of athletic individuals. Acute inflammatory changes may be present which, through irritation of the peritoneum, cause spasm of the abdominal wall. In some cases it may be necessary to give an anesthetic before a proper examination can be made. *Second;* a considerable amount of fat in the abdominal wall which may be present to such an extent as to render proper examination difficult or impossible even under an anesthetic. Pressure by the examiner's free hand on the back of the hand used in palpation aids somewhat. *Third;* the presence of free fluid in the peritoneal cavity. A sudden

stab-like pressure by the examining hand serves to dispel the fluid temporarily and allows of quick palpation of the tumor. Where the abdominal wall is rendered tense by fluid it may be necessary to tap in order to determine the presence of tumor. *Fourth;* the presence of tympanites, which if in considerable degree will interfere with a proper examination just as fluid does. *Fifth;* the presence of a distended bladder or colon, the former requiring catheterization, the latter, either catharsis or enemata before a proper examination can be made.

POSTURE FOR EXAMINATION. The patient should be upon the back with shoulders somewhat raised and thighs somewhat flexed upon the abdomen. The position should be easy and comfortable. This position refers to general abdominal examinations. In examination of certain regions the patient is placed in a partial sitting posture as for the examination of the gall-bladder and liver region; in Sims' position, right or left, for examination of kidney tumors; in Sims' position for the bimanual examination of tumors of the sigmoid.

INSPECTION. We note the cutaneous appearance: smooth, shining, tense (distention); lineae albicantes (previous long-continued distention, ascites, fat, pregnancy); linea nigra (pregnancy, chronic abdominal enlargements); relaxation (wasting diseases, carcinoma).

COLOR. Copper-colored scaly circular spots (secondary syphilis); yellow, brown or black patches of irregular size and shape (cholasma); bronze (disease or tumor of the adrenals); whether jaundiced, and the degree of jaundice: light transparent jaundice (pancreatic disease); moderate transparent jaundice (cancer of pancreas); deeper jaundice (carcinoma of liver); deep dark-brown jaundice (long-continued common duct obstruction): Chinese white (extreme hemorrhage); general pallor with yellowish-green tinge (chlorosis); blanched, smooth, waxy (progressive pernicious anemia); yellow-white cachetic (carcinoma).

The presence of *superficial veins* and their situation: Dilatation of the veins about the umbilicus shows obstruction in the portal circulation (hepatic cirrhosis or tumor); general venous engorgement indicates portal obstruction

(hepatic cirrhosis or tumor of liver) or obstruction in the inferior or superior vena cava (if inferior, abdominal tumor, if superior, mediastinal tumor); dilated lateral vein in the right mid-axillary line connecting the tributaries of the superior cava with the enlarged inferior epigastric veins (empty this vein by pressure, watch it refill, and try to ascertain the direction of the current. In obstruction of the portal vein and inferior cava the flow is upward, in obstruction of the superior vena cava the flow is downward through the azygos vein); distended veins in pubic region (obstruction, pressure or thrombosis below the liver).

The appearance of the *umbilicus:* retraction (fat abdomen); projecting (intra-abdominal pressure, ascites, pregnancy, hernia).

When *enlargement* of the abdomen is present, where is the enlargement located? It may be general (fat, ascites, meteorism); localized (tumor, cyst or distention of viscus, dilated stomach, distended bladder); enlargement of viscus (cirrhosis of liver, liver tumor and cysts, hypertrophied spleen). Ascites causes general enlargement, while ovarian cysts show enlargement according to their size in the lower part of the abdomen. Is the enlargement lateral? Is it circumscribed? If lateral, the spleen or kidneys are suggested; if circumscribed, tuberculous or pneumococcic peritonitis. A prominence around the umbilicus leads one to suspect a pancreatic cyst, if more or less rounded; while if more diffuse, a tumor of the stomach is suggested.

In a thin individual, respiration may be seen to cause the tumor to move. The absence of respiratory movement indicates peritonitis, excessive distention or painful irritative lesion; its diminution indicates ascites, meteorism or large abdominal tumor. Visible peristalsis is normally seen in a thin-walled relaxed abdomen, and is made more active by manipulation. Painful visible peristalsis means intestinal obstruction. Exaggerated strong peristaltic waves from left to right in the upper left quadrant indicate pyloric obstruction; if from right to left in the course of the colon, large intestinal obstruction.

DISTENTION if general, is usually ascites or meteorism; if epigastric and in the left upper quadrant, dilated stomach;

if central, obstruction at or about ileocecal valve; if lateral and transverse in course of colon, obstruction in course of sigmoid flexure.

PALPATION. The hands should be warm, and great gentleness should be used; much more can be felt by not causing too great pressure upon the delicate nerve endings in the fingers; also it will be possible to avoid arousing the antagonism of the abdominal muscles if palpation is gentle. At first a gentle general orientation should be made by the fingers of one hand; this will give an idea as to the muscular resistance in different parts of the abdomen; and it is from this recognition of *different degrees of muscular resistance* that the skilled diagnostician obtains much of his knowledge.

Following this orientation with one hand, the orientation with the left hand by pressure of the right hand upon the back of the hand is made, still being gentle. Having located the tumor, if it is laterally situated, bimanual examination is made with one hand placed between the ribs and the crest of the ileum posteriorly, and the other over the tumor in front; and the tumor is gently mapped out. Or if the tumor reaches to the pelvis, bimanual vaginal or rectal examination is made. Sudden stab-like pressure often serves to identify a tumor in an excessively fat or ascitic abdomen.

Tumors in the upper part of the abdomen move with respiration. Particularly is this true of gall-bladder or liver tumors and large kidney tumors, also some tumors of the stomach. Tumors below these organs do not move with respiration. The exception to the first part of this rule is that adhesions may so fix the tumor as not allow it to move.

Some tumors can be moved to different parts of the abdomen, the extent of their mobility depending upon their attachment; as for example, in tumors of the large intestine, the extent of the mobility depends upon the length of the mesentery, or its lack of involvement in the tumor process. Ovarian tumors with long pedicles have a very wide range of motion. Uterine tumors which have risen out of the true pelvis have a considerable range of lateral motion. A movable spleen has a large range of motion. On the other hand, tumors of the pylorus and kidney have smaller ranges of

motion, while retroperitoneal tumors, such as those of the pancreas and retroperitoneal sarcoma, have very little or no mobility. This is also true of encapsulated exudates.

The range of mobility is of importance. For instance, hydrops of the gall-bladder can be swung from side to side, but when it is pushed backward it springs into place again, while a tumor of the kidney, unless fixed, may be moved up and down and not laterally. One should bear in mind in movable tumors that the fixed point of the attachment is in all probability the normal point of attachment of the organ involved, and by calculating the *degree* of mobility and the *range* of mobility a fair estimate can be made as to the organ involved.

The *shape* of some tumors simulates on an exaggerated scale the shape of the organ involved. This is true of tumors of the spleen, in which case the splenic notch can be made out. It is true of tumors of the kidney, for the most part where the shape of the kidney tumor follows in a general way the shape of the kidney itself. This is also true of the enlarged gall-bladder and of diffuse myomas of the uterus. In other tumors the shape shows the character, such as multiple fibroid tumors of the uterus, and ovarian tumors which present round cystic swelling to the left or right of the uterus.

The Presence or Absence of Fluctuation. Many tumors give a faint sense of fluctuation, for example, a rapidly growing myoma of the uterus, a rapidly growing retroperitoneal sarcoma, hydatid and other cysts.

Palpation shows us the consistency of the tumor, its mobility, its size, the nature of its surface, the edges of the tumor, the presence or absence of pulsation and whether pulsation is in the tumor or transmitted.

Percussion aids in distinguishing ascites from an ovarian cyst; the former gives dullness in the flanks and tympany in the center; in the latter the opposite is true. It also aids in differentiating retroperitoneal tumors from those in contact with the abdominal wall; also in differentiating tumors with fluid contents or solid contents.

Auscultation aids in the differentiation of a pregnant uterus from other abdominal tumors; in the diagnosis of aneurysm and some hydatid cysts in which the fluid contents is not

sufficient to make the cyst tense, allowing of Santorini's booming sound.

Where doubt exists, *inflation of the stomach and colon* is of aid, particularly in the diagnosis of tumors of the stomach and large intestine, and in the differential diagnosis of retroperitoneal conditions such as tumor of the kidney, pancreas, adrenals and retroperitoneal lymph glands.

Such are the methods of diagnosis, remembering that corroborative evidence is to be obtained by chemical and microscopical analyses and the use of the roentgen ray. These accessory methods of examination may, in certain diseases, prove diagnostic. For instance, in enlarged spleen with its blood picture of secondary anemia; in enlarged spleen with plasmodium found in the blood or in borings taken from the spleen itself; the different blood pictures of acute and chronic inflammation; the roentgen-ray diagnosis of incomplete obstruction of the large intestine and the tumors of the stomach.

TUMORS OF THE STOMACH. Carcinoma is by far the most frequent. Palpable tumors of the stomach are those involving the pylorus, or extensive infiltration of the greater or lesser curvature and of the anterior surface of the stomach.

Pyloric tumors change their position with respiration and possess a marked range of passive mobility. Tumors occurring with gastroptosis or tumors which have produced marked dilatation show little respiratory mobility. Adherent tumors do not move with respiration. A pyloric tumor can be grasped and its movement by respiration prevented. Pyloric tumors with dilated stomach move to the right and downward. Inflation of the stomach in a case of tumor of the anterior wall causes the tumor to move downward. Inflation of the stomach in both pyloric tumors and tumors in the anterior wall render the tumor less distinct. Such tumors are usually felt in the epigastric and umbilical regions, but may be in the lower abdomen in cases of gastroptosis or extreme dilatation. Percussing the dilated stomach may cause peristaltic waves to pass toward the tumor.

When dilatation of the stomach is present there is visible bulging, in the majority of cases. This may be overlooked because sufficient attention is not paid to inspection, palpa-

tion, percussion and inflation. The tumor formed by a dilated stomach may be very prominent. The distention is most marked on the left side and in the umbilical and left hypochondriac regions. It becomes less after vomiting or lavage. Often the stomach contour is visible and a diagnosis can be made by this alone.

Before atony has occurred, peristaltic waves may be seen, usually in series, beginning in the left hypochondrium and extending diagonally down and across the abdomen to the midline, crossing to the right at a variable distance above the pubes and ending in a third and final contraction in the right upper portion of the umbilical region.

The ease with which peristalsis can be observed depends upon the thickness of the abdominal wall. At any time before atony has occurred these waves can be readily activated. In very thin patients the same contractions of the stomach which are observed during fluoroscopy are seen under the thin abdominal wall. As paresis of the musculature progresses peristalsis becomes less active, and local bulgings more prominent. In the early stages of pyloric obstruction these waves can be felt by the examining hand. They become stronger and more marked as the pylorus is reached.

Palpation will determine the presence of a pyloric tumor. The ease with which such a tumor can be palpated depends upon the extent of the dilatation of the stomach. A mass which is hardly palpable when the stomach is dilated will become readily so when the stomach is emptied by vomiting or lavage. What was thought in the first examination, to be a small mass, may prove, when the stomach is emptied, to be quite large.

Gastric tumors are hard, oblong, tender and usually smooth, but may be nodular.

Tumors traceable to the stomach in young persons may be carcinoma or fibromata, but in middle-aged or elderly people the tumor is carcinoma. The diagnosis rests upon (1) loss of weight, (2) secondary anemia, (3) pain, at first a heaviness after eating, later of a dull, gnawing character referred to the epigastrium; local tenderness, (4) vomiting, (5) hemorrhage, rarely fresh blood; usually coffee-ground vomitus, (6) examination of stomach contents frequently done at short inter-

vals; such examinations show absence or great reduction of hydrochloric acid and the presence of lactic acid. The constant presence of blood intimately mixed in the gastric contents is also of great importance; (7) tumor.

The above has been the teaching of the study of carcinoma in the past and at present. In my experience the early signs of carcinoma of the stomach are sudden disturbances of digestion occurring in a middle-aged individual who has previously been healthy, such errors in digestion not being traceable to cardiac or circulatory disease.

I have briefly referred to the textbook picture of carcinoma of the stomach. Operations done in such cases show deplorable results. Why? Not because the patient did not have symptoms for months before being operated upon, but because these symptoms were not properly interpreted. Here again the physician has been wrongly taught. To wait in these cases for the classical symptoms is to allow the patient to die. Too much stress has been placed on gastric analysis, too much time lost in treatment of the early disturbances of function, too much time lost waiting for a tumor to develop. As long as the present textbook teaching of carcinoma of the stomach is permitted, just so long will patients look in vain for relief. The early symptoms of slight disturbance of gastric function must be emphasized.

When a previously healthy patient past forty years presents symptoms indubitably gastric in origin, however slight these symptoms may be, which do not yield entirely to dietic and therapeutic treatment in less than a month's time, it is reasonable to suppose that a cancer is present. The physician's duty is plainly to advise an exploratory laparotomy. If the classical symptoms are waited for or a laboratory diagnosis insisted upon, the case will have passed beyond aid.

That some of these disturbances may be due to gall-bladder or appendical affections is true, but it is also true that these affections are better treated operatively, and that an exploratory operation will reveal the lesion.

Differential Diagnosis

DILATATION OF THE STOMACH. The physical signs are so characteristic that there would not seem to be ground for

error, particularly when considered in connection with the general symptoms.

The distended stomach has been mistaken for ovarian cyst. The paretic stomach of extreme dilatation may simulate ascites, particularly if the stomach is very full of fluid.

In the colon distended from tumor, the peristaltic waves are from right to left; usually the abdomen is more uniformly distended in colonic distention, while in stomach dilatation the distention is mostly on the left side; and moreover, in the former case, intestinal and not stomach symptoms are more prominent.

Inflation of the stomach, or if practicable, of the colon in combination with it, will usually assist in reaching a definite conclusion.

A dilated stomach causing a prominent tumor, as mentioned, is almost invariably accompanied by a palpable pyloric tumor. It is differentiated from atony or gastroptosis by the absence of peristaltic waves in the two latter conditions.

The stomach in Glenard's disease may be dilated, and in some cases found difficult to differentiate, but by mapping out the stomach and finding it not so enlarged as ptosed and atonic, and by palpation, recognizing the mobility of the liver and kidneys, and by inflation and the position of the ptosed colon, differentiation is made possible.

TUMOR OF THE STOMACH. This is to be differentiated from pancreatic growths, tumors of the transverse colon or duodenum, carcinoma of the gall-bladder, tumors of the left lobe of the liver and extensive ulcer of the stomach.

1. Pancreatic Growths. In elderly people with thin abdominal walls the pancreas is hard, easily palpable and may be mistaken by the inexperienced for a carcinoma of the stomach. Tumors of the head of the pancreas also simulate carcinoma of the stomach. Inflation of the colon and stomach causes the pancreatic tumor to disappear. Such tumors are not movable, as are stomach tumors. Pancreatic tumors are commonly associated with a mild degree of jaundice. There is often an accompanying glycosuria. Examination of the stools shows free fat and undigested meat fiber. Ascites is a more frequent accompaniment of pancreatic tumors. Asso-

ciated growths of the liver are common to both, but appear earlier in pancreatic growths.

2. Tumors of the Transverse Colon. Obstructive symptoms are present. Inflation of the stomach may cause the tumor to disappear; inflation of the colon makes the tumor more prominent. Gastric analysis shows no change.

3. Tumors of the Duodenum. These are so rare as not to warrant a differentiation between tumors of the duodenum and tumors of the pylorus.

4. Carcinoma of the Gall-bladder. Such tumors are not movable; they cannot be held fixed against respiratory changes in position; jaundice is usually present and a history of previous attacks of gall-bladder disease is usual.

5. Tumors of the Left Lobe of the Liver. Inflation of the stomach makes them more prominent because the left lobe of the liver lies in front of the stomach.

6. Extensive Ulceration of the Stomach. Differential diagnosis is impossible except that old chronic extensive ulcer is surmised when cachexia and rapid loss of weight are absent. Also, ulceration is more common in young individuals. Even at operation it is impossible to differentiate between some extensive ulcers of the stomach and carcinoma. I have had cases which were too extensive for resection which I supposed to be carcinoma and in which a jejunostomy was performed for feeding purposes. Several of these cases have finally completely recovered although the mass involved the entire anterior wall of the stomach.

I cite these instances here in order that exploratory laparotomy may not be denied even the most extensive case where the disease is apparently limited to the stomach itself.

TUMORS OF THE LIVER. Tumors of the liver are of rather frequent occurrence and, as a rule, are not difficult to recognize (Fig. 44). They may be classified as malignant or benign; as primary or secondary growths; as solid or cystic; as limited to a portion of the liver or involving the entire organ; or the liver itself may form the tumor.

As examples of involvement of the entire liver we may mention hypertrophic cirrhosis, amyloid liver, fatty liver, the uniform enlargement due to massive cancer, the hepatitis due to abscess of the liver, hydatid cysts, or neighboring

inflammation in the gall-bladder and ducts. Under such circumstances the organ is enlarged as a whole. We will discuss localized enlargements of the liver, and from a diagnostic standpoint consider those instances in which the liver itself forms the tumor. Such cases are not rare. The most frequent form is the *pulsating liver* found in chronic mitral disease. The insufficiency of the tricuspid valve allows each systole of the right ventricle to be communicated through the right auricle directly to the column of blood in the hepatic veins. This results in an expansile expulsion of the entire liver synchronous with the heart-beat.

FLOATING OR MOVABLE LIVER. This is either congenital or acquired. In the latter event it results from tight lacing or from the dragging upon the liver of an enlarged gall-

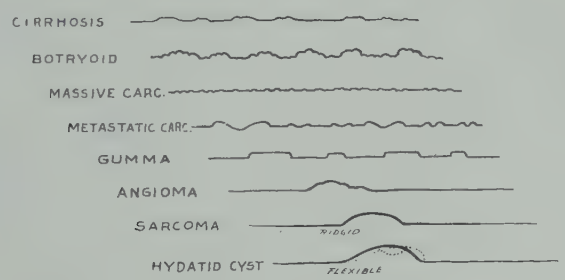


FIG. 44. Palpation lines of tumor of the liver.

bladder or a large tumor or cyst. The *congenital variety* is usually accompanied by enteroptosis. The degree of mobility depends upon the amount of lengthening of the supensory, lateral and coronary ligaments and, in rare instances, the absence of the two latter. It occurs much more frequently in women than in men. Such a liver may give rise to no inconvenience and the lesion be only discovered at the post-mortem table, or there may be such dragging pain in the hepatic region as to render the unfortunate individual's life quite unendurable. There are frequent attacks of severe pain, and jaundice may be present at times. Digestive disturbances are common. Examination will disclose a tumor of the size and shape of the liver, which in many instances can be made by manipulation to occupy the normal site. In

addition, percussion will elicit tympanitic resonance over the hepatic region. The *acquired variety* may be complete or partial. The latter is by far the more frequent. The left lobe alone may be affected. I have operated upon a case of this kind in which the etiological factor was a large echinococcus cyst springing from the inferior surface of the left lobe in the neighborhood of the transverse fissure. This had been in existence for some time, and had so dragged upon the liver that the tumor and left lobe could be moved to almost any

Fig. 45. Corset liver.

part of the abdominal cavity. Most commonly the right lobe is the one affected. This results from tight lacing and is known as *corset liver* (Fig. 45). Through pressure of the corset a portion of the right lobe is constricted. The pressure is often so severe as to cause actual deformity of the ribs themselves and this rib pressure transmitted to the liver results, in the milder cases, in scar-tissue formation in the liver itself with consequent loss of nutrition to the liver cells.

In some instances the pressure is so severe as almost to completely separate a portion of the right lobe. In other cases a deep groove or furrow marking the site of constriction divides the lower part of the liver from the upper part. In extreme cases this constriction of a portion of the liver results in changing the liver tissue into fibrous tissue so that the constricted portion consists of blood vessels, bile ducts and scar tissue. Such a condition is apt to be mistaken for a tumor. The constricted portion becomes at times so widely separated from the liver as to be only connected with it by a fibrous tissue band. Usually the part of the liver affected contains the gall-bladder, which is consequently displaced with the constricted portion of the liver. Such a condition presents at times great difficulty in diagnosis, but by careful manipulation, even in the extreme cases, the connection between the mass and liver may be made out, while in cases not so extreme the furrow can be felt and diagnosis made in that manner. The tumor, of course, unless very widely disconnected from the liver, moves with the liver during respiration. The condition is differentiated from movable kidney by turning the patient on the left side and mapping out the kidney as not a part of the mass. A movable kidney can be replaced in its proper position, but the separated corset lobe, while widely movable, is not capable of being placed in the kidney region.

In cases of enteroptosis the diagnosis is still more difficult. Most dependence will be placed upon the shape of the tumor, the tumor formed by the kidney being characteristically shaped.

From the kidney with a mesonefron, the diagnosis is quite simple, as the characteristic shape of the kidney is easily made out, though the mobility may be great.

As for renal tumors, these often have the shape of a normal kidney and of course lie behind the colon. Even when large, their dullness rarely is continuous with the liver dullness, as is the case in ordinary corset lobe. The lower edge of the renal tumor is rounded, while the corset lobe often presents irregularities. A movable liver is to be differentiated from a floating or movable kidney. Each organ has its characteristic shape.

Floating or movable liver is differentiated from tumors of the liver itself, such as carcinoma, by the increased size of the liver in the latter, as well as by the location of the liver. In carcinoma the liver is enlarged downward but still extends up to its normal level at the sixth rib, while in floating liver the size and shape of the liver is about normal but its location is abnormal; there is tympany above it.

Yet another form in which the liver may present as a tumor is botryoid liver, so called from its resemblance in shape to a bunch of grapes. Such a condition is the final result of a syphilitic hepatitis, and the changes are permanent.

ACCESSORY LOBES. Usually these are too small to attract attention and are discovered only at autopsy. Occasionally, however, they may become hypertrophied and be mistaken for new growths.

The usual causes of tumor of the liver are cancer, abscess, syphilis, hydatids and, occasionally, tuberculosis. The most common form is secondary carcinoma. Sarcoma, lymphadenoma and angioma are at times observed; more rarely still, myxoma, fibroma and atheroma. Aneurysm of the hepatic artery must be considered from a differential diagnosis standpoint. Here we have a tumor in the hepatic region which by pressure on the common bile-duct invariably causes jaundice. Pain is very severe. The diagnosis rests on the expansile pulsation and the bruit.

Secondary carcinoma follows a primary growth in the stomach in about 25 per cent of the cases, and as such is unrecognized during life. The symptoms of the primary growth are so prominent that the secondary development is overshadowed. In perhaps another 25 per cent of the cases the primary growth is sufficiently well advanced to mask the metastatic growth. In nearly all cases of cancer of the liver there is progressively increasing pain and distress in the hepatic region. There is steady decrease in weight and loss of strength which appear early in the course of the disease. This progressive emaciation is more marked in malignant disease of the liver than in malignant disease elsewhere. The organ increases rapidly in size and is tender to the touch. Gastric disturbances are frequent. Jaundice may be present early but, as a rule, it appears late and steadily increases.

The cause is found in pressure upon the common or hepatic ducts, or involvement of the ducts within the liver. Cancer is the most frequent cause of long-standing jaundice. Ascites may be present. This is due to pressure upon the portal vein or to the extension of the disease to the peritoneum. In cases which run a very rapid course there may be slight elevations of temperature from time to time. The tumor varies in size. In rare cases of long standing it may almost entirely fill the

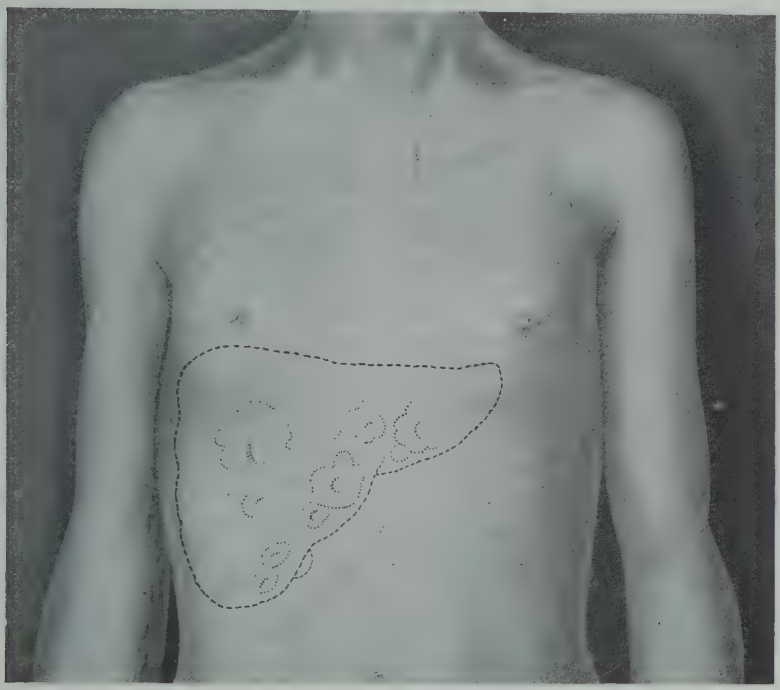


FIG. 46. Metastatic carcinoma.

abdominal cavity. Cases which involve the entire liver will present characteristic nodules upon the surface (Fig. 46). These vary in size, some being so small as to be hardly perceptible, while others are as large as an orange. They are umbilicated, hard, with rounded edges, having a depression in the center. This depression is caused by degenerative changes and subsequent cicatricial contraction occurring in the center of the mass. Occasionally these cases proceed

very slowly and are difficult of diagnosis. It is at times difficult to differentiate between carcinoma and hypertrophic cirrhosis. The presence of the nodules is characteristic. Jaundice may be present in both.

Abscess of liver may present as a palpable tumor. Such a tumor develops beneath the right costal margin or in the epigastrium. Tenderness is present unless the disease has been in existence for a considerable time. The size of the tumor rapidly increases, and as it becomes more prominent, fluctuation may be obtained. In some cases there may be no change in months. The constitutional symptoms, irregular fever, chills and sweating, together with the rapid growth and extreme tenderness of the tumor, should be sufficient to establish its character. In cases in which there is doubt, the aspirator may be used. The previous history is valuable in these cases. While dysentery may not be present, a history of it is usually obtainable. The presence or absence of ameba should be ascertained. The sallow and malarial aspect of the patient should be considered.

Syphilitic tumors are not rare, nor should they be difficult of diagnosis. They present two forms, the recent gummata and the botryoid liver, which is the final result of a syphilitic hepatitis. This form has been mentioned in the class in which the entire liver forms the tumor. It presents as an irregular, hard, knobby mass, divided by cicatricial bands. The recent gummata, however, form genuine tumors. They occur as hard nodules or flat masses of large size, presenting most frequently in the epigastrium. They differ decidedly in feeling and shape from the knobs of cancer. In many instances a clear specific history can be obtained and the diagnosis can be verified by the administration of potassium iodid. While this is unlikely to result in a total disappearance of the tumor, yet there is almost invariably a rapid marked decrease in size.

Occasionally the abdomen is opened for such tumors, when no specific history can be obtained.

At times gummata break down and find their way to the surface.

Hydatids. The chief symptoms are a sense of distress and weight in the hepatic region and the tumor. As a rule,

diagnosis is easy. The surface of the tumor is smooth and rounded (Fig. 47). Its connection with the liver is readily made out. To the touch it is tensely elastic and rarely tender. The long history, slow growth, absence of loss of weight and strength and lack of tenderness, differentiate it from cancer or abscess. Its shape and the absence of biliary symptoms rule out the gall-bladder. In cases in which the gall-bladder

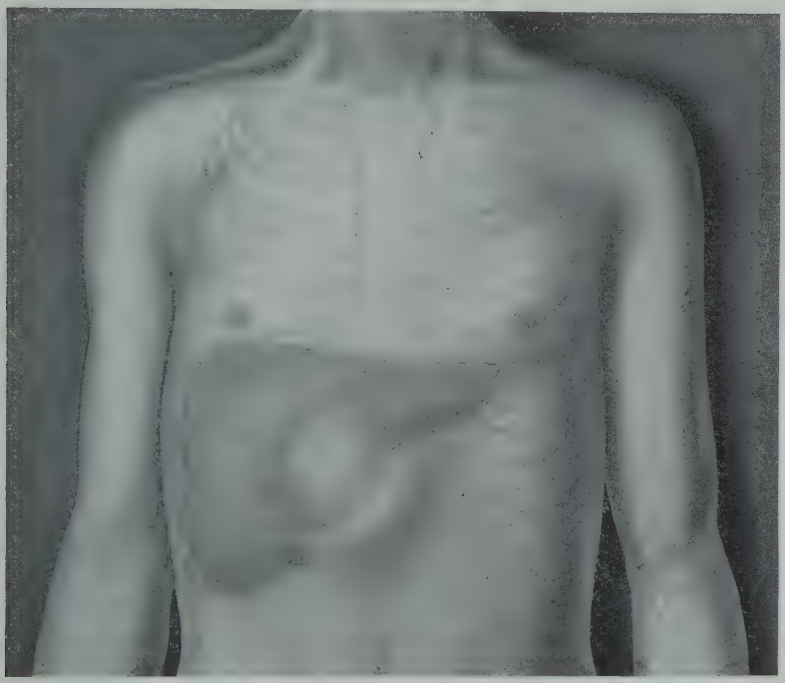


Fig. 47. Hydatid cyst.

is distended from pressure of the cyst, there will be two tumors, the enlarged gall-bladder and the cyst. The fact that the colon does not overlie the cyst rules out cystic or sarcomatous kidney or other retroperitoneal growth. In a case of my own, however, the transverse colon was so held to the anterior surface of the cyst by adhesions that there was tympanitic resonance over the cyst. The so-called hydatid fremitus, Santorini's booming sound, is present only

when the daughter cysts swim in fluid. It is caused by their impact upon each other when the cyst is percussed. It is absent in those cases in which the parent sac is too distended to allow the daughter cysts to change position, or in which there is not sufficient fluid for them to float, or in sterile cysts.

Differential diagnosis of echinococcus cyst of the liver. When the cyst is on the anterior surface it must be differentiated from *cystic disease* of the liver itself. In cystic disease the elevations are generally small and multiple, but a sufficient number of them are large enough to be felt to be cystic. This disease is so rare, however, that it is not likely to be borne in mind.

From *carcinoma,* by the cachexia and the fact that the tumors are harder and are multiple. Also in secondary carcinoma there is umbilication of the tumors.

From *abscess of the liver,* by the fact that in abscess the tumor is not so hard and tense. Usually there are fever and other septic symptoms. There may be a history of dysentery. In cases of suppurating cyst the diagnosis is difficult and at times impossible.

When the cyst projects from the lower border of the liver it is to be differentiated from a dilated gall-bladder. The dilated gall-bladder is pear-shaped and is movable. The echinococcus cyst is dome-shaped and moves only with the liver.

Diagnosis from permanent or intermittent hydronephrosis. In *intermittent* hydronephrosis by the history of alternating presence and absence of a tumor, the change associated with polyuria. In *permanent* hydronephrosis by placing the patient on the left side and outlining the hydronephrosis, which is more or less of the form of the actual kidney, projects more in the lumbar region and lies behind the colon. When doubt exists, the colon can be inflated. With the patient on the left side the echinococcus cyst is less prominent than the tumor of permanent hydronephrosis. *When the cyst is in the dome of the liver,* diagnosis must be made from pleuritic effusion. To make this differential diagnosis, exploratory puncture is necessary and an examination of the fluid. The line of dullness in echinococcus cyst is curved with the con-

vexity upward while the pleuritic effusion line of dullness is almost horizontal.

From *hydatid of the lung with pleuritic effusion* diagnosis is almost impossible except when hemoptysis and cough are present, in which event pulmonary involvement is suspected.

From *subphrenic abscess.* The diagnosis is made by the history and by the fever. *X*-ray examination may aid in showing the shape of the cyst. Exploratory puncture is of value.

Sarcomata. Primary sarcoma is very rare. Few authentic cases have been reported. I have seen one case in a child.

Adenomata. These are also uncommon. They may remain quiescent for years and finally undergo malignant change. The diagnosis can be made only by the long existence of the tumor and the absence of severe symptoms.

Angiomata. Angioma is the most frequently observed variety of the benign tumors of the liver. There are two forms, the congenital and a form which appears later in life. The former is of exceedingly rapid growth and rapidly fatal. The form which appears later in life occurs mostly in males of sixty years and upwards, rarely before forty. There may be a single tumor, but more often they are multiple. They rarely attain to a large size and are almost invariably separated from the liver substance by a fibrous capsule. They rarely become large enough to cause symptoms. I recall one case.

Fibromata and other tumors are rare.

Tumor as a symptom in gall-bladder disease. There are cases of cholecystitis in which the inflammation causes a swelling of the gall-bladder so that it forms a more or less palpable tumor. Each attack of inflammation increases the exudate in the cavity of the gall-bladder so that it becomes further enlarged and finally reaches a size which can be palpated. A normal gall-bladder cannot be palpated. The only time at which the gall-bladder can be palpated is when the cystic duct is closed, and even then only under special conditions. In some cases the gall-bladder is situated deep under and in the right lobe. When such a gall-bladder becomes acutely inflamed and enlarges, the surrounding liver also becomes swollen and tender. In such a case, palpation

of the gall-bladder is difficult and many times impossible. The diagnosis is made by the *local* enlargement of the liver, the area of tenderness and the depth of the tenderness.

An enlarged gall-bladder is pear-shaped. It forms an oval tumor which moves up and down with the liver on respiration. Such a gall-bladder can almost always be discovered by percussion and palpation. If the gall-bladder is attached to the peritoneum its movements are restricted to a certain extent according to the character of the adhesions. When adhesions are not present a characteristic of the gall-bladder tumor is that it can be shifted laterally and mesially. An enlarged gall-bladder swings on the cystic duct. This is the more evident, the more loosely the gall-bladder is attached to the liver, and also in cases in which the right lobe of the liver itself is movable. At times there is a real mesentery of the gall-bladder, and in such cases the tumor can be moved well over into the hypochondrium and epigastrium.

As a rule, the shape of an enlarged gall-bladder is so characteristic that when once established the condition in which it was found is not easily forgotten.

Mistakes in diagnosis, however, have been made. The condition has been confused with right cystic kidney, right movable kidney (Fig. 48), tumor of the ascending colon or of the hepatic flexure, and hydatid cyst of the liver, more often than at first would seem possible. Quite often a right movable kidney is confused with hydrops of the gall-bladder. A gall-bladder the seat of hydrops lies directly behind the abdominal wall and points downward toward the umbilicus, forming a pear-shaped tumor. It is movable laterally and mesially to a greater or less extent according to the mobility of the right lobe of the liver, and also according to the length of the gall-bladder attachment to the liver itself. When pressed away from the abdominal wall it returns to its normal position in relation with the abdominal wall. Movable kidney, on the other hand, moves in an up-and-down direction away from the diaphragm. Its lateral mobility is not so marked. It is more deeply situated, and it can be returned to its normal site with a characteristic snap. Its form is characteristic. In very stout patients, when doubt exists as to differ-

entiation, distention of the colon with air may be employed, and possibly ureteral catheterization with *x*-ray. Congenital wandering kidney can be differentiated by bringing it to a part of the abdominal cavity where its shape can be determined.

There are times when an enlarged gall-bladder cannot be felt. This is so when it is covered entirely by the liver, or

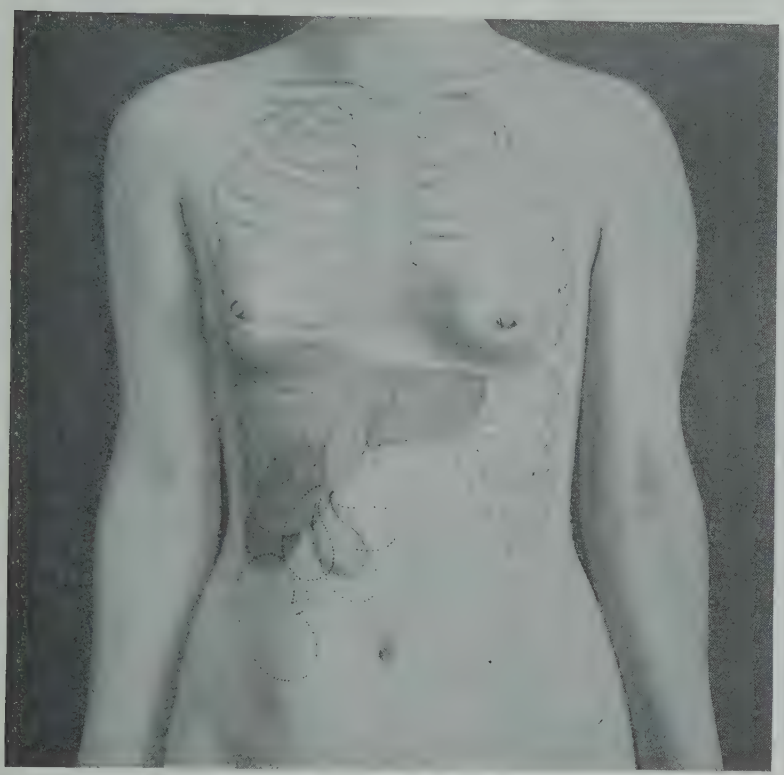


FIG. 48. Differential diagnosis between hydrops of gall-bladder and movable kidney.

when it is so covered by adhesions to the omentum, stomach and colon as to lie behind these organs; also in very fat people who have strong and rigid abdominal muscles, and when rigidity from inflammation is present.

In the examination of cases to determine the increase in the size of the gall-bladder, great gentleness must be used.

A rough examination increases the rigidity so that nothing can be felt. In rigid cases a hot bath will often relax the abdominal wall and render palpation easier. An injection of morphin will help in this regard. At times it is only under an anesthetic that the tumor can be made out. In making the abdominal examination it is necessary that the hand be warm. In abdominal examinations for any lesion this is to be borne in mind, and also the fact that fingers are to be trained to transmit to the brain the picture of the lesion that the fingers feel. When one first makes abdominal examinations the finger transmits to the brain only what it feels, as for instance the roundness of an hydatid cyst, or the resistance or lack of resistance from an acute or chronic inflammation; but as time goes on and one becomes more educated in abdominal diagnosis, the finger transmits a picture of an hydatid cyst, etc.; in other words, its true pathology. By thinking along these lines, the finger can be trained to a very much greater degree than when only the terms "a hard mass" or "a soft mass" or "some fluctuating mass" are used to describe what is felt. In time one comes to say: "I feel an acute cholecystitis with closure of the cystic duct." "I feel an hydatid cyst attached to the liver."

Diagnosis between Serous and Gangrenous Cholecystitis. A tender tumor of the gall-bladder is felt. Is this a serous cholecystitis, or is it purulent or gangrenous? If serous, sensitiveness to pressure will decrease very soon, but if purulent or gangrenous, sensitiveness to pressure will *not* decrease. It is often necessary to make several examinations with some period of time between, to determine whether a tender gall-bladder tumor is serous cholecystitis or a purulent or gangrenous cholecystitis.

Of course this is a very important differentiation to make, as a serous cholecystitis can be allowed to subside before operation is done, while a purulent or gangrenous cholecystitis should be relieved by drainage at once.

In some cases with very slight inflammation, a gall-bladder tumor is easily palpated, but in severe infection palpation is rendered more difficult on account of the rigidity of the abdominal muscles. On several occasions when examining patients during acute attacks I have found a gall-bladder

tumor to disappear in the course of the examination and have felt the gall-bladder empty itself under the examining fingers. In such cases one concludes that the case does not need an immediate operation, but should be watched for a few days, so that when the acute symptoms have subsided the operation can be more safely performed.

Many times in a very fat person there is found a large gall-bladder which could have been easily palpable had the abdominal muscles not been so rigid. In the most severe cases of acute cholecystitis there is felt a diffuse, painful resistance. By gentle manipulation of this area of resistance the size of the gall-bladder can be mapped out. I must say, however, in many cases it is not possible to map out the gall-bladder. We must be guided by the area of tenderness.

The decision as to whether a serous or gangrenous cholecystitis is present is not always possible to make in one examination. If the pulse is rapid, temperature high and general condition poor, and if there is characteristic sensitive resistance of the gall-bladder region and collapse, then the indications are that the case is severe gangrenous cholecystitis, and an immediate operation is advisable. These cases simulate gangrenous appendicitis, acute pancreatitis and acute intestinal obstruction.

Rarely does a gangrenous cholecystitis perforate. Perforation is diagnosed when the patient who has a gall-bladder forming a tumor with temperature, severe pain and a general sick look, suddenly has the pain lessen somewhat, shows some shock, then a rise of temperature and general lowering of the resistance accompanied by an *increase* in the area of *tenderness* and *rigidity*. The condition is somewhat analogous to a perforated appendix, and those familiar with this condition should have no difficulty in making a diagnosis of gall-bladder perforation.

Differential Diagnosis between Gall-bladder Disease and Right Movable Kidney. Hydrops of the gall-bladder stretches the neck of the gall-bladder to such an extent as to allow a wide range of motion from side to side. A point well worth remembering in the diagnosis of tumors of the abdomen is the demonstration of the extent and direction of range of motion. If the fixed normal point of the attachment of the

organ involved be borne in mind, this range in extent of movement of the tumor is usually readily traceable to the proper organ. The attachment to the liver does not permit of the hydrops being pushed into the kidney region. In the case of the enlargement known as Riedel's lobe it is possible to differentiate the kidney through palpation by turning the patient on the left side. In cases in which the liver is prolapsed, differentiation of the kidney is somewhat more difficult, as in these cases a gall-bladder the site of hydrops is pushed to the right so that in position it more closely resembles a prolapse of the kidney. In this condition, however, it is quite easy to palpate the edge of the liver and to recognize it as such, using the direction in which the edge of the liver lies, its lowest point on the right side rising abruptly upward to the left and along this line, to map out a gall-bladder the site of hydrops.

In those cases in which a gall-bladder has a mesentery of its own, hydrops is not so easy to differentiate from movable right kidney and requires careful examination before it can be decided whether there is a movable kidney or a hydrops. If by any chance a right movable kidney shows a cyst of the lower pole anteriorly placed, the difficulties of differential diagnosis are increased. It should be borne in mind that a considerable mobility of the right kidney exists normally in women, and it may well be that a right movable kidney can be coexistent with an enlarged gall-bladder. In these cases also it is possible to separate the kidney from the gall-bladder by placing the patient on the left side. By so doing the liver is pushed forward, and in this manner the fingers can be placed between the kidney and the liver. Careful palpation will reveal the true condition.

A person suffering from a right movable kidney can have the same symptoms as one suffering from gall-bladder disease: colic, or stagnation of bile in the gall-bladder with or without jaundice. The common duct is involved mechanically and the symptoms can occur without the formation of stones.

The differential diagnosis between renal colic and gallstone colic is not difficult if the patient's history is carefully considered. A point in differential diagnosis is to turn the

patient on the left side so that the liver is pushed forward; then by palpation the lower edge of the liver is recognized and the gall bladder region and these parts are found free from pressure pain, whereas in the lumbar region there is a circumscribed pressure pain limited to the right kidney region. In this connection one must remember there are cases of gall-bladder disease in which the pain is felt only in the back and in which there is no pain whatever in the gall-bladder region; but these are exceptions, and happen very infrequently. In the further differentiation of renal colic from gall-stone colic a careful urinalysis will aid materially, as well as a combined radiographic study and ureteral catheterization.

THE DIAGNOSIS AND TREATMENT OF THE MORE COMMON RECTOCOLONIC DISEASES*

SAMUEL G. GANT, M.D.

EXAMINATION. There is no excuse today for not diagnosing anorectal affections, for they may be inspected through the proctoscope and the character of the feces detected by chemical, microscopic and macroscopic examination. Moreover, the extent and connection of rectal diseases with neighboring organs can be determined by digital exploration, bimanual palpation, fluoroscopic examinations and roentgenograms.

Preparation of Patient. The common practice of purging patients and deluging the colon before rectal operations entails annoyance and infection, since the field of operation will be bathed with fluid feces, which delays the work, contaminates the wound, soils the dressings and makes the patient uncomfortable afterward.

For fistula, hemorrhoidal, fissures, polypoidal and other minor operations, but a few moments are required to prepare the patient. This is done by the injection of a glassful of soapsuds into the rectum to bring away solid feces, after which the mucosa is mopped over with peroxide of hydrogen or other antiseptic: procedures which forestall many annoyances. When the bowel is to be amputated or resected for rectal procidentia, stricture, tuberculosis or cancer, three or four days' preparation is required and the gastrointestinal tract is first cleansed by catharsis and colonic irrigations of ichthyol 1 per cent, and then tied up with an opiate or an astringent. This insures a dry field of operation. The parts are never shaved except where sutures are used and primary union anticipated, because the hair stubs cause great annoyance.

Morphia, ¼ gr., is administered fifteen minutes before the operation to forestall postoperative pain and, should suffering continue, the dose is repeated in half an hour.

*This lecture was delivered on Apr. 27, 1923.

Postoperative Treatment. General surgeons are divided in their method of controlling the stools after rectal operations. Some purge the patient, liquefy the feces and thus keep him busy and miserable, while others administer an opiate and tie up the bowel for days. These procedures are to be condemned because the former insures continuous irrigation and favors infection through soiling the field with fluid feces, and the latter leads to excruciating pain and tearing open of the wound when accumulated dry and nodular fecal masses are evacuated.

The writer keeps his patients on a regular diet, and does not order a laxative unless the stools are hard, when he prescribes mineral oil, fruit or other laxatives in small doses to soften but not to liquefy them; then at one sitting semisolid feces are evacuated without pain or injury.

The former practice of cauterizing and packing wounds no longer obtains, for they were pernicious procedures, responsible for many unhealed sores and much fecal incontinence. For cleansing wounds, the swab and cotton moistened in an antiseptic are substituted for copious irrigation; for the latter leaves the rectum filled with the solution that dribbles out, soils the dressing and keeps the patient uncomfortable. When healing is tardy the wound is satisfactorily stimulated by placing in it a gauze pledget moistened in a solution of silver nitrate 6 per cent, ichthyol 10 per cent or balsam of Peru 20 per cent; but when the tissues are irritable and rebel against stimulants, methylene blue 10 per cent is found to be soothing and effective.

Pain from topical applications can be minimized by applications of eucaine or cocaine 10 per cent; but all suffering can be prevented by injecting eucaine ($\frac{1}{8}$ per cent) beneath the lesion before it is treated.

LIMITATIONS AND TECHNIQUE OF LOCAL ANESTHESIA IN RECTOCOLONIC OPERATIONS

In a series of cases, including anal, rectal, colonic and abdominal operations, different forms of anesthesia are necessary and the operator must choose between *general, spinal, sacral, regional* or *infiltration* anesthesia, according to his preference and the requirements of the case.

In the writer's practice, alarming bleeding has occurred ten times more frequently following operations performed under general than under infiltration anesthesia, and post-operative pain has been more frequent and severe after the former than after the latter.

Since 1900, about 80 per cent of the writer's private and clinical anorectal and a considerable number of abdominal operations, have been performed with satisfactory results under infiltration anesthesia induced by sterile water, normal salt solution, eucaine, novocaine and other anesthetic agents. He has operated five thousand times under local anesthesia without a death, dangerous toxic manifestation or other serious complication.

In the beginning, owing to imperfect technique and employment of solutions of different strengths, patients occasionally squirmed or complained of pain; and in a few instances general anesthesia was substituted for local analgesia. These complications have rarely been encountered in the last fifteen years, since the present technique has been employed.

The scope of infiltration anesthesia has, in recent times, been greatly extended; and physicians, surgeons and proctologists who do not perfect their technique and employ it largely to the exclusion of general narcosis, do not deserve the patronage of patients afflicted with minor anorectal affections.

Infiltration anesthesia possesses many desirable features, but it also has its limitations. The writer never employs it except when he has ascertained beforehand what is to be done; and he discards the method for spinal or gas-ether anesthesia for extensive deep operations, in which an unknown amount of cutting may be required.

General anesthesia is also preferred in minor affections complicated by a high or a more serious rectal disease, also when tissues are necrotic or there is an ulcer or fistulous opening preventing retention in and desensitization of the tissues by the anesthetizing solution.

To aid the reader in forming an opinion concerning indications for general and local anesthesia in this class of work the writer has grouped affections most frequently encountered about the terminal bowel as follows:

1. Anorectal diseases *operable* under local anesthesia.
2. Anorectal diseases *inoperable* under local anesthesia.

ANORECTAL DISEASES OPERABLE UNDER LOCAL ANESTHESIA

Internal protruding, bleeding, capillary, combination; external, cutaneous and thrombotic hemorrhoids; fissure-in-ano, ulcers of the anal canal, single and multiple, superficial, long and short fistulae; procidentia-recti (first and second degree), cryptitis, inflamed and hypertrophied rectal papillae, hypertrophied rectal valves, marginal, submucous and ischiorectal abscesses; polyps, strictures (below the peritoneal attachments), foreign bodies (imbedded beneath the mucosa or skin), constipation incident to sphincteralgia, hypertrophy of the levator-ani muscle or narrow anal canal, intrarectal diverticula and cysts, incipient anal epithelioma, perianal cysts, tumors and condylomata; fecal impaction, pruritus ani, sacral dimples, dermoids and fistulae, fecal incontinence, repair of anorectal injuries and sequelae to operations for congenital malformations.

The writer has several times performed under infiltration anesthesia, exploratory celiotomy, colostomy, appendicostomy, cecostomy, ileostomy, sigmoidopexy, colopexy, uterine suspension and other abdominal operations, including breaking up of adhesions, straightening Lane's kinks, division of Jackson's membranes and colo-plication.

He has also relieved acute intestinal obstruction by opening the abdomen and draining the bowel under local anesthesia, and has several times performed the secondary operations of excising the gut, opening the cecum or small intestine, or amputating the appendix in connection with colostomy, cecostomy, ileostomy or appendicostomy.

In some, the operation was entirely painless, while in other instances the patient suffered slightly on incision of the fascia, peritoneum or pulling upon the mesentery.

Experiments with infiltration anesthesia have convinced the writer that *general* (gas-ether or gas-oxygen) is preferable to *local* anesthesia for deep and abdominal operations, in which considerable handling of the intestines or viscera is required.

ANORECTAL DISEASES INOPERABLE UNDER LOCAL ANESTHESIA

Cancer (barring beginning anal epithelioma), extensive perirectal and ischiorectal abscesses, deep burrowing complicated horseshoe, perirectal rectourethral, rectovaginal, and vesicorectal fistulae, high polyps, strictures, ulcers, cysts, nonmalignant tumors and imbedded foreign bodies; submucous abscesses and fistulae in the upper rectum and any disease or injury requiring amputation or resection of the lower bowel.

General is also preferable to local anesthesia for procedures and plastic operations that require extensive stitching in and about the rectum, except those employed in the radical treatment of fistulae and hemorrhoids; and is desirable when a minor is complicated by a more serious rectal affection, also in case of lesions that cannot be brought into view for operation, and deep abdominal or operations requiring traction upon the mesentery.

A study of the diseases included in the above list shows that approximately 80 per cent of anorectal affections are located within easy reach and are capable of being operated upon under local anesthesia.

EQUIPMENT, AGENTS AND METHODS

Posture. The exaggerated knee-chest, lithotomy or left Sims' posture may be employed, but the writer prefers the latter because it is convenient for him and comfortable for the patient during operations upon the buttocks, anus or lower rectum.

Syringe. A metal syringe having a Gant goose-neck is preferable to a glass syringe, which breaks easily or leaks under pressure during skin, fistula and stricture operations, in which tissues are dense. Small needles are objectionable because they readily break or become blocked by corrosion or ingredients in the solution.

Local Anesthetics. Anesthetizing agents save the patient considerable suffering in both palliative and operative treatment of anorectal diseases.

PALLIATIVE TREATMENT

Suffering incident to examination, instrumentation, cauterization and treatment of sensitive individuals may be prevented or minimized by placing a pledget of cotton soaked in a 10 per cent solution of eucaine, cocaine or novocaine upon lesions in the mucosa or perianal skin, and letting it rest there for a few moments. These agents do not induce complete anesthesia here, as in the throat, but effectively desensitize the part when injected into or beneath the sensitive area in a solution of ⅛ of 1 per cent.

Orthoform, alypin, anesthesin and analgen, possessing analgesic and healing properties, markedly diminish pain and muscular irritability incident to walking, sitting, defecation and treatment of local lesions, when employed in a powder, suppository, solution or ointment, and they deserve a place in the armamentarium of the proctologist.

Eucaine and cocaine solutions are also useful for mitigating and arresting unbearable itching—pruritus ani—when the skin is raw.

OPERATIVE TREATMENT

Numerous agents have been employed for anorectal operations, viz., ether spray, liquid air, ethyl chloride, eucaine, cocaine, stovaine, novocaine, tropacocaine, sterile water, normal saline solution, quinin and urea, hemesia and apothesine.

Ether-Spray, *Ethyl-Chloride* and *Liquid Air*. These agents deaden or prevent cutting pain by freezing and benumbing the tissues; but they are unsatisfactory because they induce acute initial and postoperative suffering, are often followed by sloughing, and when effective induce only superficial anesthesia. Thus their employment is limited to light incisions in the mucosa and skin.

Solutions. Eucaine, cocaine, stovaine novocaine, quinin and urea apothesine and tropacocaine are solutions that have their respective adherents and have been successfully employed in varying strengths to produce spinal, paravertebral, regional and local anesthesia for anorectal, pelvic and abdominal operations.

These drugs, particularly cocaine, often produce annoying or alarming toxic manifestations when used in a strong (3 to 10 per cent) solution; but in a weak solution they are effective and seldom disturb the patient when injected in small amounts. The majority of operators employ solutions of from ½ of 1 to 2 per cent, which is stronger than required to produce complete desensitization of tissues.

Extensive experiments made in 1900 convinced me that the anesthetizing action of the solution is due both to the contained chemical and to the distention of tissues with compression of the nerves; for I observed that infiltration anesthesia failed or was incomplete when part of the fluid escaped through a needle puncture, necrotic tissue, ulcer or fistulous opening. I also demonstrated, by hundreds of anorectal operations, performed under sterile water and normal saline infiltration, that analgesia immediately follows when tissues are distended with the fluid until glassy white, indicating complete anesthesia.

Repeated experiments with eucaine, cocaine, novocaine, stovaine, tropacocaine and like agents satisfied the writer that eucaine and novocaine are the anesthetics of choice. During the last fifteen years he has operated five thousand times without an accident and without failing to obtain complete anesthesia with eucaine (⅛ of 1 per cent solution); and such patients were able to walk from the operating room to their beds, rarely exhibiting toxic manifestations, irrespective of the amount of anesthetic employed.

The addition of adrenalin (1–1000) extends duration of analgesia and lessens hemorrhage during operation, but the writer seldom employs it, because blood vessels, temporarily constricted, later relax and bleed freely; and he prefers that hemorrhage should take place during operation so that it may be immediately controlled.

Sterile Water and Normal Salt Solution. During the period of 1900 to 1905 I published a series of papers giving the technique of distention anesthesia induced by the injection of sterile water and normal saline solution, and reported several hundred anorectal operations successfully performed under these agents. Subsequently sterile water was aban-

doned for eucaine anesthesia because the initial injection induced slight pain.

Quinin and Urea Hydrochloride. This anesthetic is effective, non-toxic, suitable for long and for short operations, and minimizes postoperative pain. The solution when injected causes greater pain than does eucaine or novocaine, and its use requires minutes longer to produce complete anesthesia.

Quinin and urea is not desirable for skin operations, since it causes induration or sloughing of the integument and delays healing. It induces anesthesia when employed in a solution of ¼ to 1 per cent. The writer sometimes employs this agent in the clinic or office when operating on patients desiring to return home.

Apothesine, which is practically non-toxic, has been successfully employed in a solution of ½ to 1 per cent, but it requires a longer time to induce analgesia than does eucaine or novocaine.

Varying sensibilities of different patients have nothing to do with results obtained, nor does success of the anesthetization depend entirely upon the anesthesia employed; for good or bad results depend on whether or not the anesthetizing agent is retained in the tissues, and whether a sufficient quantity of the solution is injected to cause a round or linear whitish swelling over the part to be incised.

The amount of sterile water or anesthetci employed vraise from a few drops to one or more ounces, depending on resistance of tissues and extent of the operation. For example, four to six drops suffice for the removal of hypertrophide papillae, inflamed crypts and small polyps; a half drop for external cutaneous thrombotic piles and one drachm or more for fissure, medium-sized or large internal hemorrhoids, from half to an ounce or more for fistulectomy, low strictures, Gant's pruritus, procidentia-recti operations and division of the sphincter.

Before giving anesthesia the writer tells all patients that the preliminary injection will cause discomfort, but that the operation will not; otherwise they squirm at the needle puncture.

In skin operations, after the initial injection, consisting of one or two drops, is made intercutaneously, forming a bleb, the solution is slowly deposited subcutaneously into the operative field until tissues are distended glassy white, an invariable indication of analgesia.

To obtain effective anesthesia it is essential that the operator avoid multiple unnecessary needle punctures, fissures, ulcers and fistulous tracts; otherwise pressure action of the solution upon nerves is lost, owing to escape of the fluid through the openings.

The operator must also work quickly, since the solution, when properly prepared and injected, induces anesthesia within a few seconds, and analgesia lasts only a few moments.

I have heard surgeons say they objected to infiltration anesthesia in anorectal operations because patients squirmed, fainted or complained of severe pain. These annoyances are rarely encountered by the writer, whose results have been universally satisfactory. I am certain that unsatisfactory results reported by some operators would have been avoided had they understood the technique of infiltration anesthesia, employed weak in preference to strong solutions of eucaine or novocaine, and used tact with their patients.

It might be observed at this point, that, contrary to the theories of some prophets, both the number of my patients and the size of fees received from them have materially increased since I began employing local in preference to general anesthesia.

The writer is fully convinced that when proctologists and surgeons become proficient in the technique, and employ local anesthesia to the extent called for, they will be able to perform many anorectal operations formerly considered impossible except under general narcosis. Thus the elimination of advertising quacks from this field of surgery will be greatly facilitated.

Hemorrhoids are exposed or extruded for infiltration with the aid of a slide speculum, or by tipping them out with the end of a Gant speculum as the instrument is withdrawn, everting anal margins with fingers, withdrawing them by cupping, administering a small enema and extruding them by

pulling on tampons introduced into the rectum through a proctoscope.

In completing the operation, in case of copious oozing or spurting vessels, hemorrhage is controlled by a twisted gauze plug inserted through a proctoscope which, when withdrawn, leaves the plug projecting through the anus.

POSTOPERATIVE TREATMENT

Following an average hemorrhoidal or fissure operation and excision of papillae, crypts and polyps, the patient is permitted to leave the hospital in two or three days, or when he can comfortably come to the office in a taxi for dressings. These patients are allowed a regular diet, and normal stools are induced with fruit or a mild laxative.

Postoperative pain is practically nil in intrarectal, but is severe in extensive skin, operations, and lasts from half to two hours, unless controlled by morphin administered hypodermically in quarter-grain doses as often as required.

LOCAL ANESTHESIA IN ABDOMINAL OPERATIONS

Infiltration anesthesia is not so universally satisfactory for abdominal as for anorectal and surface operations, where it is effective. The procedure is contraindicated in this class of operations, unless the operator knows beforehand just what is to be done; and local anesthesia is not suitable for many deep abdominal and pelvic lesions, extraction of tumors when viscera are matted together by adhesions and where there are complicating diseases within the abdomen.

The writer has performed with local anesthesia, 55 abdominal operations, including appendicostomies, cecostomies, enterostomies, colostomies, cecopexies, cecoplications, colotomy, sigmoidopexies and operations for Jackson's membranes, Lane's kinks, adhesions and angulations, some of which were performed under sterile water anesthesia and reported in a series of papers published between 1902–1905.

The majority of the above-named procedures are adaptable to local anesthesia, because deep exploration is often unnecessary, pulling upon sensitive mesentery is seldom required, and pinching, cutting and suturing of the unesthetized gut causes little or no pain. Discomfort from the

majority of these operations is avoided by the expert in local anesthesia, who carefully prepares and injects his solution slowly and cautiously, and infiltrates each abdominal layer (from skin to peritoneum) *separately*. This usually requires about three minutes each for the skin, subcutaneous structures, muscles, fascia and peritoneum.

The principles in abdominal infiltration are, in the main, the same as those employed in local anesthesia for anorectal operations already described, and the analgesic action of the anesthetic is due both to distention pressure on nerves and to the chemical contained in the solution.

The writer prescribes morphin (gr. $\frac{1}{4}$) and scopolamine (gr. $\frac{1}{200}$) one hour preceding, and repeats the dose fifteen minutes before abdominal local anesthesia operations in robust individuals; this is to blunt consciousness and quiet apprehension in nervous patients.

Analgesia is more effective when the anesthetic is used liberally and the solution slowly diffused into the operative field. Satisfactory surgical analgesia is produced with a solution of eucaine ($\frac{1}{8}$), novocaine ($\frac{1}{2}$ of 1), or apothesine (1 per cent), in combination with adrenalin (1–1000), which quickens and lengthens action of the anesthetic, with but little danger of causing toxic manifestations even when the solution is used freely.

COMMON DISEASES OF THE BREAST*

JOHN E. JENNINGS, M.D.

OUR subject has been plainly indicated, and this is fortunate, because it gives us a little margin. The diseases of the breast as they are described in the textbooks, are many. A varied number of pathological conditions, surgical and otherwise, are described, but, fortunately, most of those described are rare. We have, however, a certain number of diseases to cover, and it will be well, I think, for us first to get a general view of the diseases as they occur. Some we shall not be able even to name. Many we shall pass over briefly.

CHART 1

ANOMALIES OF THE BREAST

1. Amastia.
2. Polymastia.
3. Polythelia.
4. Malformations of the nipple.
5. Atrophy
6. Hypertrophy.
7. Gynecomastia.

AMASTIA

When an arrest of development begins before the third month in utero, it results in complete absence of the breast. This may be unilateral or bilateral, and is very rare. When an arrest of development occurs later, it causes an incomplete development, less rare, but also uncommon; and slight differences in size and development are relatively quite common. The nipple may be absent on one or both sides and is subject to minor forms of maldevelopment, such as inversion, depression, flattening and so forth.

*This lecture was delivered on Oct. 5, 1923.

Polymastia

More common than amastia is the presence of one or more accessory breasts in a rudimentary condition. These supernumerary glands follow the milk line of the embryo, which runs from the axilla to the groin, and is well seen in the fetal pig. In the human embryo,there are seven potential rudiments of breasts on each side, of which three are above and three below the normal pair. Supernumerary breasts are apt to occur in pairs, but may be unilateral. More than three-fourths of all cases are found in the site of the fifth pair, just inside and below the normal pair. They are not uncommon at the site of the second pair in the axilla. They may be the seat of infection, inflammation, new growths and even carcinoma, which must be carefully distinguished as an aberrant mammary growth and treated accordingly. They have been observed in other situations where they are called mammae erraticae: on the face, the ear, the hip, the thigh, the buttock, the back, the labia.

Supernumerary nipples are relatively common and are found more often in the male than in the female, in the proportion of two to one. They may be multiple in one breast or found in various situations. Their treatment is removal, if convenience or cosmetic reasons indicate it, or if the aberrant structure is recognized as the site of disease.

Hypertrophy

This is also rare, and occurs in the following forms:

1. In precociously developed children, the breasts remain large.

2. In the male a condition known as gynecomastia occasionally occurs, in which one or both breasts may enlarge, and may even secrete milk. This is often associated with atrophy of the testicles.

3. Diffuse hypertrophy occurs in young girls, or at an early pregnancy. One or both breasts are involved in a growth that does not include the nipple, which is flattened over the mass. Large veins pass over the surface, localized edema increases, and infection may occur. The breasts are tense and boggy, or may contain nodular masses. Their

size may be enormous, cases of those weighing fifty pounds being on record.

The histology of the condition varies and may consist of:

(*a*) A diffuse fibroma.

(*b*) An hypertrophy of the glandular elements as well; or

(*c*) Adenomatous masses scattered through a diffuse fibromatosis.

FIG. 49. Lipoma of the breast.

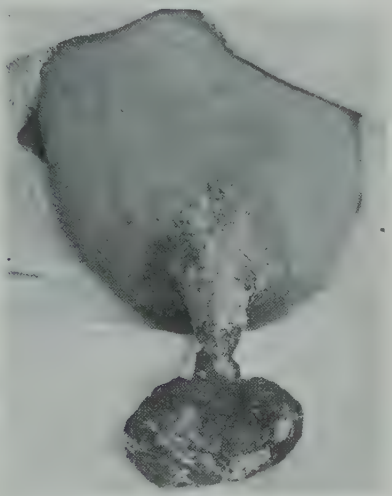


FIG. 50. Pedunculated papilloma of the nipple (gangrenous).

The treatment in the virginal form consists of removal of the breast by amputation. When the condition occurs in pregnant women, there is a probability of subsequent diminution of the growth.

CHART II

INFECTIONS OF THE BREAST

1. Pyogenic (staphylococcic).
   - A. Mastitis of the new born.
   - B. Mastitis of puberty.
   - C. Mastitis of lactation.
   - D. Mastitis in the male breast.
2. Tuberculosis of the breast.
3. Syphilis of the breast.
4. Typhoid mastitis.
5. Actinomycosis of the breast.
6. Sporotrichosis of the breast.

Inflammation

Acute mastitis may occur in both sexes and at any age.

The predisposing causes are congestion and trauma. The exciting cause is infection. Soon after birth, at the end of the first week, the mammary glands of the infant very frequently become engorged, swollen and tender, and secrete a little colostrum or milky-colored fluid. The reason for this is still under debate. One theory, and perhaps the most plausible, is that the hormone in the mother's milk, associated with her own lactation, affects the child and creates in it an activity of the mammary gland until its economy becomes accustomed to it.

Occasionally, when such breasts are treated roughly and handled by the fingers of unclean or careless attendants, or when (a primitive custom) they are sucked, they may become

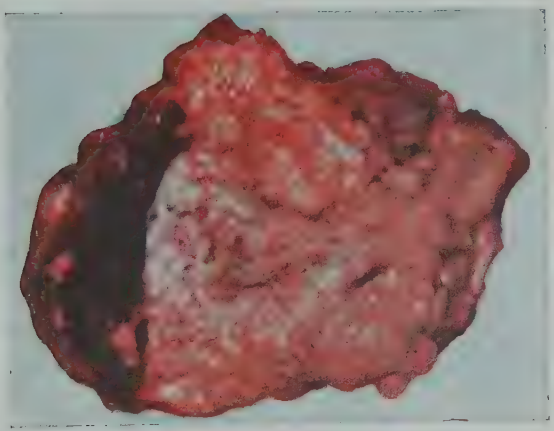
Fig. 51. Sub-mammary lipoma.

inflamed and infected. The infection is usually the common skin organism, the staphylococcus albus. Occasionally, abscesses may form and require incision, which is rather rare.

The Mastitis of Puberty. At the time of puberty, an engorgement appears, varying greatly in the individual. It is frequently symmetrical and, as a rule, entirely harmless in itself; usually unnoticed, and when noticed, amounts to very little. Sometimes it creates quite a disturbance, and there is infection and abscess formation. These are very similar to the deeper abscesses that form in the lactating breast, and require the same treatment—incision—and the

PLATE I.

1



2

1. Cancer cyst of the breast.
2. Scirrhous carcinoma of the breast.

same surgical care. This will be discussed in connection with that disease.

The Mastitis of Pregnancy. Today, with Dr. Pomeroy, I saw for the first time, an abscess which I am satisfied is one of this type, occurring before delivery. Dr. Pomeroy said he had never seen one. They are described in the literature, but if Dr. Pomeroy never saw one they must be rare indeed.

FIG. 52. Acute mastitis of right breast.

Mastitis in the Male Breast. This is to be noted as a rare curiosity.

Mastitis of Lactation. Most common is the acute mastitis of lactation. Here a caked breast, caused by retention of the milk, appears. It involves a part of the breast only, usually a single lobe, which is knotted and indurated. There is a good deal of tenderness and some pain. The constitutional symptoms are slight. Recovery is prompt when the retention is relieved by proper massage, the use of a breast pump and a good supporting bandage. If not relieved, the condition goes on speedily to infection, and pathogenic mastitis in the form of abscess or diffuse suppuration occurs.

Chart III

Suppuration in the Lactating Breast

1. Intra-mammary infection.
   A. Lobar.
   B. Disseminated.
   C. Diffuse phlegmonous.
2. Mammary abscess.
   A. Subcutaneous abscess.
   B. Intra-mammary abscess.
   C. Retro-mammary abscess.

Fig. 53. Diffuse carcinomatosis of the left breast in pregnancy.

Abscess of the Breast

This is noted in three forms:

1. Superficial. In the subcutaneous tissue of the areola a small nodule, the size of a hazel-nut, appears on one side of the nipple, at the margin of the areola. It is hard, tender, red or purple, and becomes soft and fluctuating. A small incision, radiating from the nipple, and light packing are all that are required.

2. Infection of the gland proper usually follows a fissure or a crack in the nipple after a caked breast.

The most common is that in which multiple abscesses in different parts of the breast communicate with one another by irregular channels. They should be opened by as many incisions as are necessary for good drainage, the incisions radiating from the nipple, and not extending inside the edge of the areola.

FIG. 54. Medullary carcinoma of the right breast.

3. The Submammary. This is a rarer form of suppuration that occurs as an involvement of the large lymph spaces lying behind the gland, between it and the deep fascia covering the pectoral muscle—the so-called submammary bursa of Velpeau. The gland is lifted up by the pus collection beneath it. The pain is continuous, dull and throbbing. The breast is hot, tense and of a deep red or purple color. Fluctuation may not be present until late on account of the tension under which the pus is confined.

The constitutional symptoms are more severe if the case is neglected: the pus will burrow to the surface, most often

at the upper and inner quadrant of the breast, where its substance is thinnest, or it may present on the axillary side.

The treatment is:

(*a*) Prophylactic. Proper cleanliness and care of the nipple in nursing women. Suitable attention to a caked breast.

(*b*) Early incision, which is best made under the lower periphery of the breast, widely draining the abscess, after having broken down with the finger the compartments and

FIG. 55. Rapidly growing carcinoma of the right breast in lactation.

trabeculae which may be found in the abscess cavity. Hot, wet dressings should be applied for several days.

(*c*) Diffuse suppurative inflammation. Occasionally, as a result of infection carried by the ducts, multiple foci of suppuration develop in the gland itself. Many of these, if seen and properly treated early by multiple incision, recover with a scarred but useful breast. Others, neglected, with poor drainage, sinuses and a rapidly extending infection, will require excision of the breast.

Chronic Abscess

Sometimes in lactation a circumscribed part of the breast becomes painful, hard and tender, but without treatment the infection does not go on to involvement of the skin; the symptoms subside, but the mass remains. If this is peripheral it is easily recognized, but when it occurs, as it is most apt to, in the submammary bursa, beneath the nipple, it will give a sensation of a solid tumor. Such chronic abscesses long neglected, may simulate a tumor, and the differential

Fig. 56. Carcinoma of the male breast.

diagnosis may be possible only by section. Examination of such a mass shows a firm or elastic and tender tumor in or beneath the breast. The nipple is likely to be retracted, and the skin about it, not adherent. The mass, as a rule, will be adherent to the deeper parts. It is to be distinguished from cancer, with which it is easily confused, on account of the retraction of the nipple. In cases of short duration (less than three months) and in women under forty, drainage may be used, but the breast may be lifted up by an incision underneath it, and the mass removed from below.

Tuberculosis of the Breast

This is a rarer condition. One hundred cases were collected in the work of Deaver and McFarland, and a few

reports have occurred in the literature since. No cases are reported before puberty, and the number decreases after the menopause. The oldest reported case is fifty-three. It is a disease of the maturity of the gland. The predisposing causes are lactation and previous inflammation.

There are two forms:

1. The nodular, in which one or more nodules are found deep in the parenchyma. It may remain the same size for

FIG. 57. Involvement of the axillary skin in carcinoma.

years, but usually it softens within a year. Painless in the early stages, it is not adherent to the muscle; the skin is involved by ulceration and late fistula formation. When the nodules are central, the ducts will be dragged on, the nipple retracted and cancer simulated.

2. The confluent form. The onset is more acute, there is greater enlargement, pain occurs early and caseation and fistula formation soon appear. This form, which usually occurs during lactation, suggests infection by way of the milk ducts.

The diagnosis is from abscess and carcinoma. In both forms of tuberculosis the axillary glands are involved and may overshadow the disease in the breast, which is likely to develop in the outer quadrant of the breast, and in some cases has seemed to follow axillary gland disease.

FIG. 58. Carcinoma of the breast invading the axillary skin.

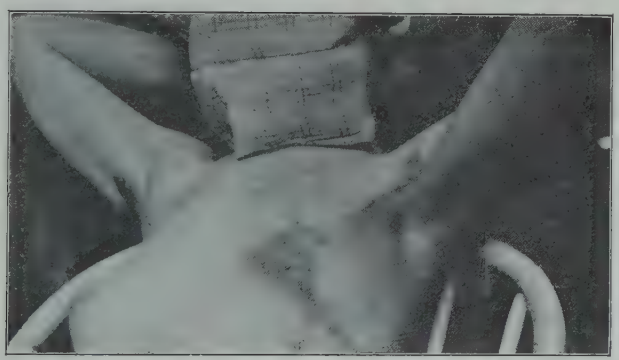


FIG. 59. Cicatrix of habitual operation limiting motion of the arm.

The treatment is:

(*a*) Removal of the breast.

(*b*) General treatment of tuberculosis.

SYPHILIS OF THE BREAST AND TYPHOID MASTITIS

These may be mentioned as occasional, but very rare conditions.

CHART IV

BREAST TUMORS

Benign	Non-indigenous tumors	Malignant	Carcinoma
	Lipoma		Adenocarcinoma
	Enchondroma		Duct carcinoma
	Myxoma		Acinar carcinoma
	Angioma		Scirrhous carcinoma
	Endothelioma		Medullary carcinoma
	Liomyoma		Colloid carcinoma
	Fibro-epithelial tumors		Paget's disease
	Fibrous type		Epithelioma
	Epithelial type		

When we come to consider the tumors of the breast, we find once more a number of conditions, most of which are comparatively rare. We will consider first the benign tumors, remembering that these occur mostly in young women, although occasionally they may be found in patients more advanced in years. Lipoma, angioma and papilloma of the skin are occasional and may be called accidental, as they do not involve the true mammary tissues. The pure fibroma is rare, and pure adenoma rarer still, but fibroadenoma is the common benign tumor and, next to carcinoma, is the most frequent tumor of the breast. It occurs (mostly in young women under forty) as a hard, painless growth, which is not tender, is freely movable, may be multiple, usually situated at the periphery of the gland and is superficial and not adherent to the skin; and the radius of the breast is not affected. It is smooth, rounded, lobulated, and varies from the size of a pinhead to that of a child's head, although rarely not so large. It may grow rapidly in pregnancy as the result of cyst formation or when associated with sarcomatous change.

The treatment should be removal of the tumor, which may be readily accomplished by a plastic operation, in which the breast is lifted up and the tumor removed from its under surface, thus placing the scar underneath the fold of the breast, where it will be hidden. The removed tumor should be examined and its nature determined, or checked by the study of frozen sections, so that if malignant disease is present, an immediate radical removal may be performed.

The practice of removing small doubtful tumors under a local anesthetic, with a delay in the histological examination

of the tumor, cannot be too strongly condemned. By the time the malignant character of the tumor is discovered an extensive dissemination of the disease may have occurred.

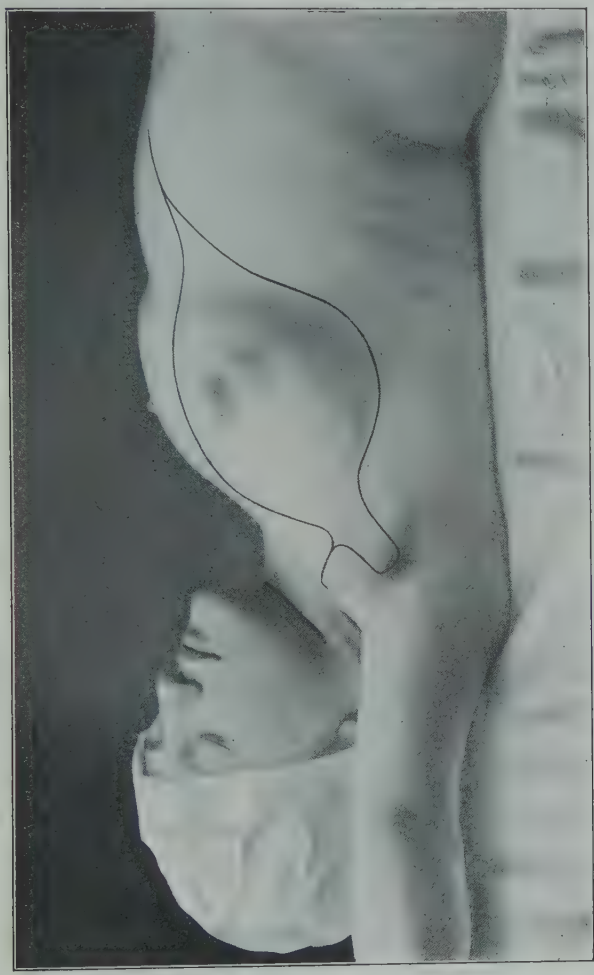


Fig. 60. Line of incision.

Malignant Tumors of the Breast

Sarcoma, one of the rarer tumors of the breast, constituting about 4 per cent of all breast tumors, is probably most common between thirty and forty, but is also seen in the aged.

There are two forms. One originates in the fibrous tissue around the acini, like the fibroadenomata, and includes in its growth the epithelial structures of the gland, which

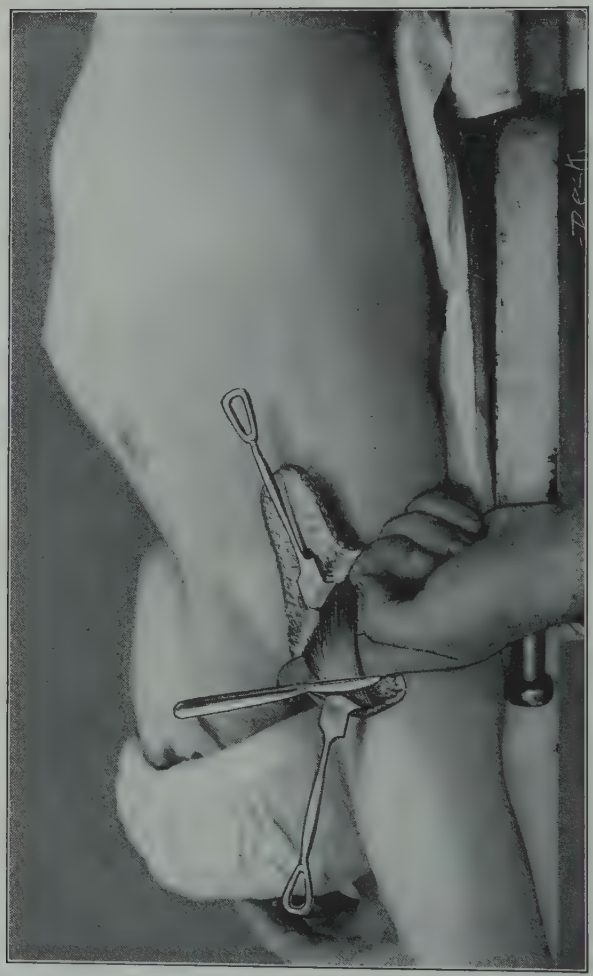


Fig. 61. Dividing the pectoralis major.

become cystic, thus resembling fibroadenoma of the cystic type, from which it often originates. This is a spindle- and a round-celled type.

The second variety is solid and originates in the true connective-tissue stroma of the gland. The sarcoma begins as a small, painless nodule, which may remain stationary for

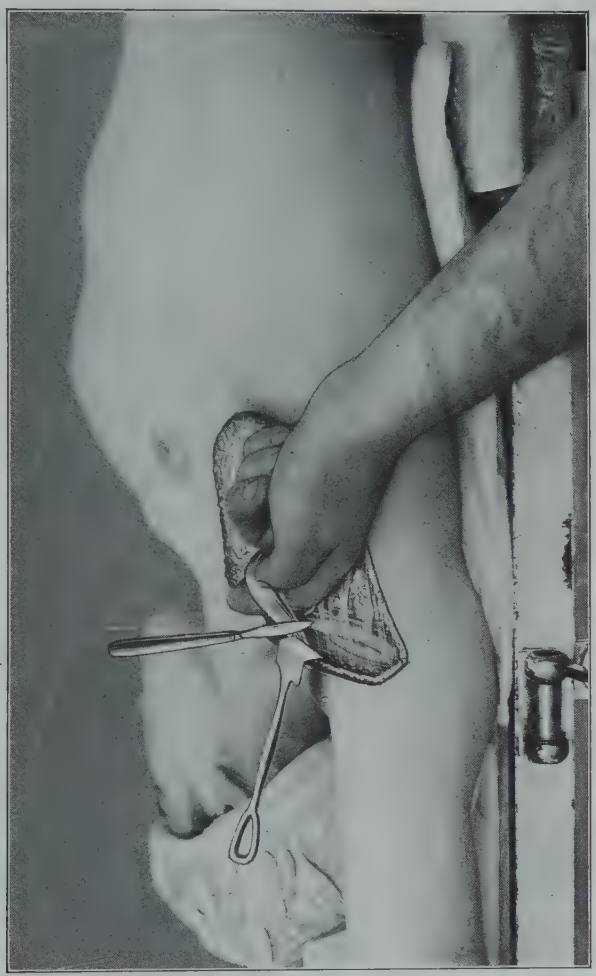


Fig. 62. Dividing the pectoralis minor.

a long time, or may slowly grow. Rapid growth then begins, the capsule which has enveloped the tumor is broken through, the gland is invaded, and a tumor of large size is formed. The skin becomes involved, is blue and discolored. Large

veins course over it, it becomes edematous, and a fungating mass breaks through. The tumor is not adherent to the muscle, and the axillary glands are not involved. The tumors may be of very large size.

Fig. 63. Clearing the axilla.

If the condition is recognized early and prompt operation performed, the prognosis for cure is good—from 50 to 75 per cent.

Carcinoma

Carcinoma of the breast is a common disease. It constitutes about 40 per cent of all cancers and probably one-half of all breast cases seen by the surgeon. Ninety-five per

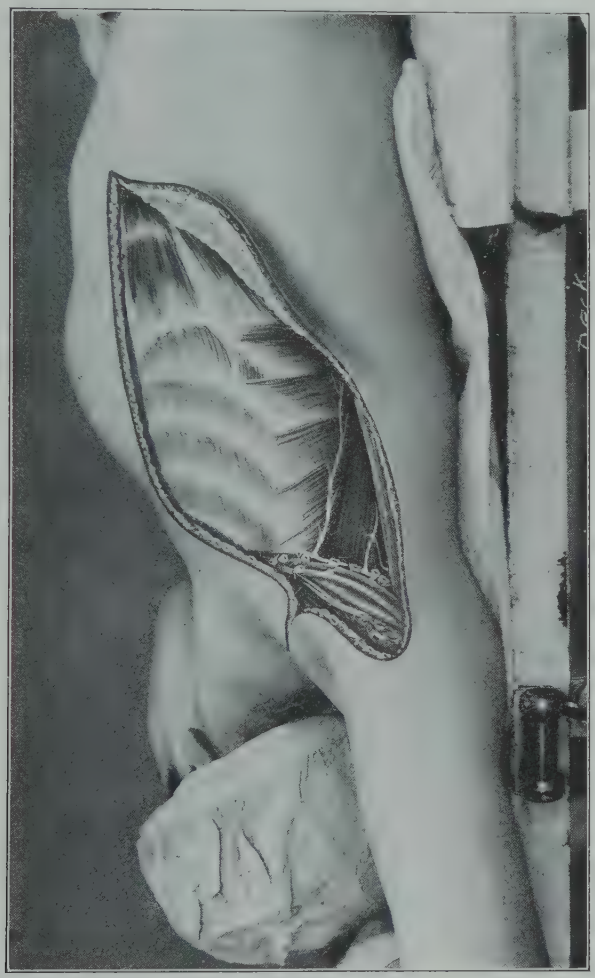


Fig. 64. Breast removed.

cent of all breast tumors in patients over fifty are found to be carcinoma. It is rare under thirty; it is not common under forty; it is most frequent from forty to sixty. In a certain

number of cases it follows chronic mastitis, and there is apparently a certain hereditary predisposition. It is more common in women who have suckled children. Occurring in the beginning as a small and localized growth, it spreads

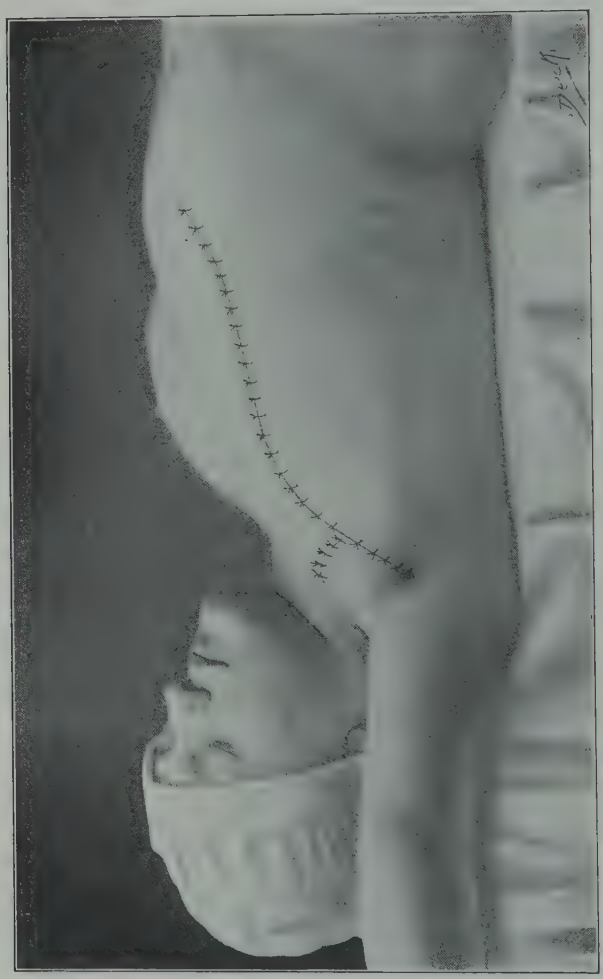


Fig. 65. Suture line.

by permeation of the lymphatic vessels slowly, centrifugally, and by continuity from its seat of origin in a way that recalls the mode of spread of erysipelas, though in a deeper plane. The lymphatics of the breast are most abundant.

The smaller channels pass from the periphery of the gland to the great lymphatic spaces behind the nipple. From this center the lymphatic channels ramify, passing to the axilla upward toward the clavicle and toward the subclavian vessels inward with the perforating branches of the internal mammary, and outward along the intercostal vessels. There are several groups of lymph glands that interrupt the lymph stream of the larger ducts. These glands are:

1. Along the axillary blood-vessels, from base to apex.
2. Posteriorly, beneath the edge of the latissimus dorsi.

FIG. 66. Unrestricted use of the arm three weeks after operation.

3. Anteriorly, along the edge of the pectoralis major.
4. A group just below the clavicle, between the pectoralis major and minor muscles; and
5. Smaller glands interrupt the course of the lymph vessels running into the mediastinum with the branches of the internal mammary artery.

The small lymph vessels ramify and form an anastomosing network in the skin and on the deep fascia. It is on the deeper plane that cancer makes its early spread.

Varieties of Cancer

1. Scirrhous and medullary. Irregular clefts in the connective-tissue framework are filled with epithelial cells of the growth. The scirrhus has more connective tissue. The medullary has more cellular elements. The scirrhus, by far the most frequent, the slowest in local growth, is characterized by hardness, nodularity and a tendency to contract. It begins as a small nodule in the breast, remains stationary for some time, then begins to grow more rapidly. As it grows peripherally it contracts centrally, then contraction drags on the nipple, causing retraction, and on the skin, causing dimpling. The pain is slight or absent. As it extends, it involves the underlying muscle, which becomes adherent. The lymph channels and the deep fascia are invaded. The axillary lymphatic glands are involved. In the later stages, the skin itself is involved, and the characteristic pigskin or orange-skin appearance makes itself evident. The tumor is often small, while the distant growth in the lymphatics or internal organs is most marked. Eventually, the liver, the lungs or the bones are invaded. Ulceration of the skin, which is a late symptom, is slow in its progress and forms a sluggish, depressed and often quite painless sore. There are no fungating masses. As the growth advances, the axillary glands compress the vessels, the supraclavicular glands enlarge, there is pain in the arm from pressure on the brachial plexus. There is edema of the arm. The course may extend over a period of from three to twenty years.

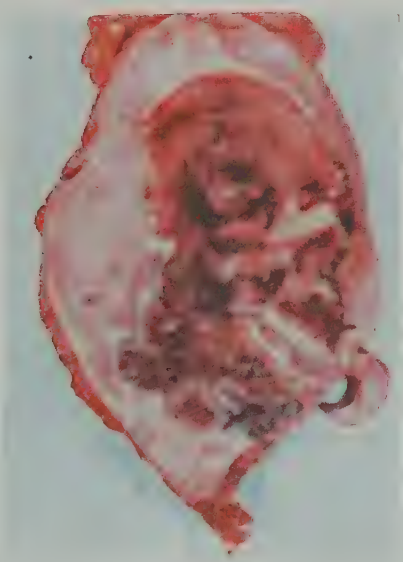
Medullary carcinoma. Two to four per cent of the cases occur in younger patients—from forty to fifty years. The disease begins as a movable nodular mass, not encapsulated, but well marked off from the surrounding structures, does not retract the nipple or the skin, and forms large masses of fungating tissue like sarcoma. The mode of extension is the same as that of scirrhus, but more rapid. The course is short and is measured by months, as a rule.

Adenocarcinoma

The growths show a tendency to produce glandular structures in the connective tissue. Carcinomata originating

PLATE II.

1



2

1. Chronic mastitis.
2. Adenocarcinoma of the breast.

In cases in which a small tumor without axillary involvement exists, and in which a diagnosis can surely be made only at the time of operation, 90 per cent of cures may be hoped for.

In the later cases with skin and axillary gland involvement, palliation and delay are all that can be expected.

It is highly desirable that the education of the public should be further developed so that every woman may know the necessity for early counsel on any mammary mass. The idea that cancer of the breast is a hopeless disease prevents a woman who has noticed a lump in her breast from seeking advice until the actual state of pain drives her to her physician. If she could be brought to understand that a benign condition in the breast does not entail the sacrifice of the organ, and that her only hope, in case the disease is carcinoma, depends upon prompt and radical action, her course would be more reasonable and more fortunate than it too often is today.

DIABETES AND INSULIN*

LOUIS C. JOHNSON, M.D.

AS Joslin so joyously remarked in a recent treatise on diabetes mellitus, "Insulin is here"; and since iletin, or insulin Lilly may now be obtained by any physician merely by writing the ordinary prescription, it seemed to me that I should do a far greater service to present, in a definite and concrete manner, the clinical problems as they appear in our patients, than if I should give a summary of the experimental work of the last two years, and leave you to go your way experimenting on your own responsibility, as we have had to do. For so great has been the publicity of the whole matter that sooner or later your patients will force you to decide whether or not you will use iletin. If the ten great killing diseases of mankind met in council, how must diabetes mellitus chuckle over her new-found greatness, and gloat over cancer, or tuberculosis, or pneumonia, the great "Captain of the Men of Death." For she has basked in the limelight for two seasons now. It is my fond hope that the whole business will turn us all to thinking in metabolic terms, as we care for our patients, and thus make the foundation for the therapeutic procedures to be employed in a given case. Undoubtedly the endocrines have mainly to do with metabolism; and if one but considers the romancing that has gone on in that field, it is easily seen how great is the need for careful clinical work and observation, and how, with a basis of a working knowledge of metabolism, such data will lead us to rational consideration and use of these glands and their products.

We have to do today with insulin, or iletin, the internal secretion of the pancreas, a deficiency of which in an individual induces the condition called diabetes mellitus.

It has been estimated that there are a million diabetics in the United States, and that new ones are being discovered or appearing faster than the old ones die. Whether they be

*This lecture was delivered on Nov. 23, 1923.

more or less in number by a few hundred thousand, it is evident that they cannot all be cared for by a few men specializing in this one phase of metabolic disorders, and that the majority of them must go untreated or undiscovered, if the general practitioner turns from them or treats them lightly.

While to know all about diabetes, its beginnings, its complications and its end, may well be a life's work and more, it by no means follows that one must devote one's entire time to the subject to be able to manage diabetics intelligently and well.

It is a great comfort to remember that so great a clinician as Joslin still insists that most diabetics are mild; and since mild diabetics have always died of complications, and since insulin will tide a diabetic over in almost any emergency, it remains for us to watch them carefully and keep them out of trouble, with or without the use of iletin, as the case may be.

When I was young the first reading-book in school was called a primer. A primer for diabetics and doctors ought to start something like this:

1 kilogram = 2.2 lb.,
1 gm. of fat = 9 calories,
1 gm. of carbohydrate = 4 calories,
1 gm. of protein = 4 calories.

Man requires daily from 25 to 30 calories per kilogram of body weight.

Nitrogen is the one food element which man must have. If he does not have nitrogen in his diet, he dies.

Protein supplies nitrogen. Man must have from ¾ to 1 gm. of protein daily per kilogram of body weight.

Children require more. They are growing, and must not only replace tissue, but build up tissue; so that they require from 1½ to 2 gms. of protein per kilogram of body weight.

The bulk of calories in any diet will be supplied from fats.

Fats are richest in calories. Most diabetics will tolerate from 2 to 2½ gms. of fat for every required gram of protein.

The remainder of the diet is to be made up of carbohydrates, and should be supplied according to the requirements as you figure them. As an example: an accountant,

who is not doing hard physical labor, weighs 132 lbs. Let us figure his diet.

$$132 \div 2.2 = 60 \text{ kilograms of body weight}$$

He will require 1 gm. of protein per kilogram of body weight, so that the protein will equal $1 \times 60 \times 4 = 240$ calories.

He will be able to tolerate in fats probably 2½ gms. for every gram of protein which you give, so that fat equals $60 \times 2\frac{1}{2} = 150 \times 9 = 1350$ calories, a total of 1590 calories. To a man weighing 60 kilograms, if we are generous, we will give 1800 calories, 60 times 30. We have already figured out for him 1590 calories, so that we have yet to supply him 210 calories, and we are going to give him that in carbohydrates; so we will divide that by 4, and that means we are going to give him somewhere in the neighborhood of 52½ gms. of carbohydrate. His diet, therefore, will consist of:

Protein	60 gms.
Fat	150 gms.
COH	52½ gms.

Such a diet, for any diabetic who has not severe acidosis or who has no infection, is, I believe, perfectly safe.

Now, in the second reader, we ought to learn that 100 gms. of fat, when they are burned in the body, will yield glucose 10 gms. and fatty acid 90 gms.; 100 gms. of protein will yield 58 gms. of glucose after it is metabolized, and there will also be produced 46 gms. of fatty acid; 100 gms. of carbohydrates will yield 100 gms. of glucose and will yield no fatty acid.

We must consider these things in balancing a diet for a diabetic, and bear in mind those substances that are ketogenetic or anti-ketogenetic; that is, carbohydrate forms no fatty acid and is therefore anti-ketogenetic. The ketone or acetone bodies are incomplete oxidation products of fatty acids. These bring about acidosis in the diabetic, and their production has been taken into consideration in the diet that we have worked out for you by giving an amount of carbohydrate sufficiently large to insure complete oxidation of all fatty acids.

We ought to distinguish between acidosis and ketosis. If a man starves, that is, if he is given no food, he will begin, as

soon as he uses up the glycogen which has been stored up in his tissues, to show acetone in his urine; and he may show diacetic acid. The reason for this is that his metabolism is carried on at such a low rate, due to lack of carbohydrate, that he is burning his own body tissues; and as a result of that he is getting incomplete oxidation of his fatty acids, the substances we have mentioned. This is ketosis. If these substances accumulate faster than they can be excreted, we have acidosis, or diminished bicarbonate of the blood.

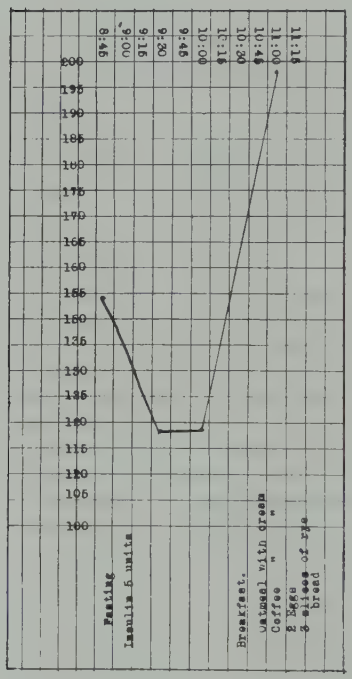


Fig. 67.

I have prepared some graphic studies of cases of diabetics. Instead of presenting a mass of purely laboratory data, I have plotted out in dots and lines the behavior of these people in the terms of ordinary clinical procedures, such as any physician might employ. It is hoped that the demonstration of these simple examinations and observations, with certain exceptions, will assist you in carrying on your own cases.

Figure 67 is an illustration which always gives me a thrill or two, because it represents our first human experiment with insulin at the Long Island College Hospital, in July 1922. Having read of the wonderful things which were being done in Toronto, we asked for directions for making the extract; and after we had begun at the beginning and acquired the glands and run them through, a process of ten days or two weeks for the first batch, we injected what we had obtained into a rabbit and it seemed to be what we thought we ought to have—insulin. We asked a patient to come to see us one Sunday morning in the laboratory. He was a stevedore who worked six days a week, a very mild

diabetic, who had no sugar in his urine at night and a fraction of a per cent by day. It was impossible to get him to adhere to a proper diet. He came in at 8:45 in the morning; his blood sugar at that time was 155 mg. per 100 c.c. of blood. Within ten minutes he was given what I estimated as about 5 units of iletin as we know it. Within a half hour his blood sugar determination was made and it had dropped to 118 mg. per 100 c.c. of blood. I asked him continually how he felt. He said he hadn't noticed any change, so I kept him a half hour longer and did another blood sugar and it had fallen no lower; but he was a little restless and so I sent him out to get some breakfast. I told him to take some oatmeal with cream, coffee with cream, 2 eggs and 3 slices of bread, and then to come back. He returned, and his blood-sugar determination at 11:00 o'clock was just under 200 mg. per 100 c.c. This illustration shows very definitely that in a diabetic individual, insulin will cause a prompt fall in the blood-sugar level, that this fall can be stopped and a rise in blood sugar brought about by taking carbohydrates, that the rise is fairly prompt, and in a diabetic is considerable. I would like to suggest also that the blood-sugar determination is a momentary reading of the individual's glucose content in the blood.

It might be well to consider for a moment the value of blood-sugar determination in following diabetics. If I were to have one single determination on a diabetic, provided he were a known diabetic, I would not ask for blood sugar. With the average practitioner, the first thing he thinks about is having a blood-sugar determination; but this, to be of value to any one, should take into consideration the time relation of the determination to the meal, and also the character of the diet which that individual is using; that is, an individual may be in coma or very near coma with a blood sugar as low as 250 mg. On the other hand, he may have a blood sugar of 600 mg. or more and be in no immediate danger of acidosis or coma. Blood-sugar determinations are, however, valuable in following cases of diabetes; that is, we believe that any case of diabetes, to be well treated, ought to have a urine absolutely free from sugar. There ought to be no acetone or diacetic acid in the urine. His

blood sugar ought to be, in most cases, within normal limits; the only exceptions being the cases of individuals who have had diabetes for many years, who show a small amount of sugar in the urine, who have arterial changes. I have found it inadvisable to bring the blood-sugar level of those individuals within the normal range as we know it, that is, from 80 to 120 mg. I think in these cases the blood sugar need not be within normal limits.

There are certain types of potential diabetics, or individuals who present the one great symptom of diabetes, glycosuria, in whom it is necessary to make very careful blood-sugar studies to determine whether or not they have diabetes mellitus. For all practical purposes, however, any individual who shows continual sugar in the urine on a diet with an ordinary intake of carbohydrate, should be considered as a diabetic individual; and his carbohydrates, having in view the next five or ten years of his life, should be restricted.

There is a type of diabetes described as renal diabetes, in which the disturbance is a renal one and there is no inability to utilize carbohydrates. Blood-chemistry determination will sort out such an individual.

In treating diabetics who are in difficulties and who are getting large amounts of iletin, blood-chemistry figures are absolutely essential. This is because the fall in blood sugar is so rapid that there may be a drop within the interval between the time of excretion and the time of examination of the urine, in which symptoms of hypoglycemia may occur; and blood chemistry is the one thing which will indicate this.

You have doubtless heard about insulin shock. We all know that if the blood sugar falls below 50 mg., these individuals are very uncomfortable. They become pale, restless and hungry, and perspire, and if the fall is great enough they will become unconscious. This condition, as you know, is absolutely relieved by the administration of glucose. With my first patients I used to try to give them insulin shock while in the hospital.

Figure 68 represents the blood-sugar curve of an individual with a moderate dose of iletin, but with starvation diet. He was a man twenty-seven years old. He was given 20

units of iletin one-half hour before breakfast and in one-half hour after breakfast (at 9:00 o'clock) his blood sugar had not fallen at all. However, at 10:30 there was a drop to 200 mg. and at 11:30 it had fallen to 126 mg. His breakfast was one lean in carbohydrates, relatively high in fats, but with the whole caloric amount small. He had dinner at 12:00 o'clock. At 2:30 the blood sugar, in response to his meal, rose to 166 mg. At 8:00 o'clock, in spite of the supper which he had, his blood sugar was as low as 118 mg. The following morning it had risen to 166 mg. This will represent to you

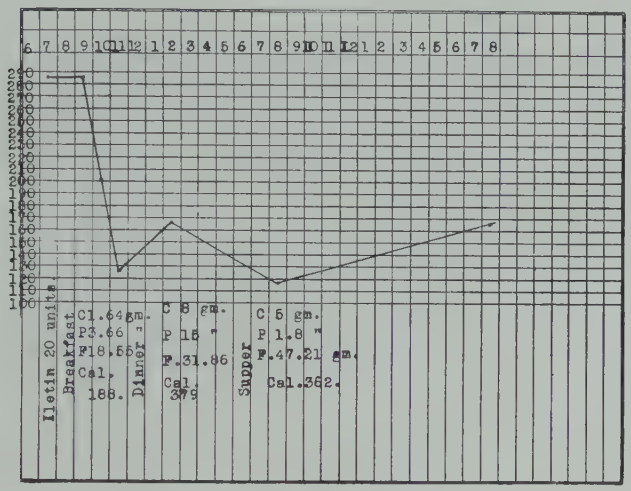


Fig. 68.

the fluctuations which take place in any diabetic's blood during the twenty-four hours. There is always a fall after the injection of iletin; there is always a rise after a meal, and those things must be borne in mind. There are certain diabetics whom it seems necessary to de-sugarize in a comparatively short time—individuals who have a pruritus or uncomfortable sensation in various parts of the body. They are very uncomfortable, and, as we have known for years, as soon as we reduce their hyperglycemia the nervous symptoms subside rapidly. There are other times when we want to de-sugarize a patient rapidly, as in case of furuncles, infections, etc.

However, I wish to show that the old method of de-sugarizing patients is still as useful as formerly, and I will explain briefly the scheme in making out the charts. The following ones are very much like Figure 69. I have aimed to record for you the things which you yourselves can do with any of your diabetics. You will observe that the top scheme represents a tracing or record of the patient's urine sugar output daily. The next represents his acetone and diacetic acid;

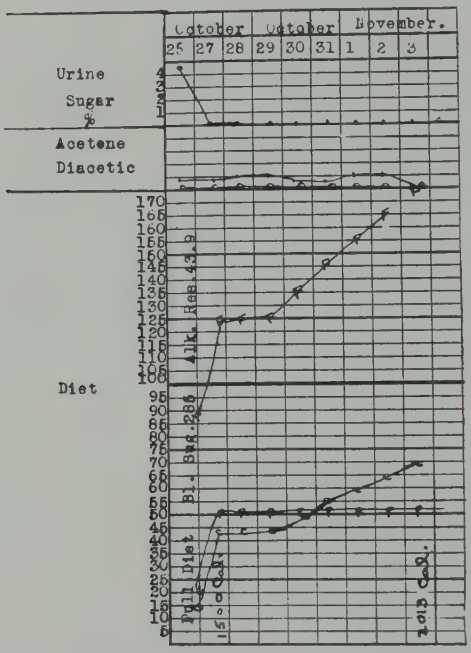


FIG. 69.

and the scheme of record we employ is 1, 2, 3 or 4 crosses, whereas below I have charted out on a rather large scale this man's diet. You will see that on the 25th he had a diet containing about 15 gms. of carbohydrates, 21 gms. of protein and 87 gms. of fat. That diet represents about 1000 calories, and you will see that very promptly, in one twenty-four-hour period, he lost all the sugar from the urine. His acetone and diacetic acid excretion were not remarkable. He had traces of acetone. He had no diacetic acid. The

curve for diacetic acid is represented by circles at the lower level; that for acetone, by dots above. This represents the curve for acetone. Then we began the following day a diet calculated as the one which I worked out for you. We gave him a maintenance diet of protein, the required amount of fats and carbohydrates, a total of 1500 calories. With that diet he would be able to go about his work and feel perfectly well and maintain his weight, or gain in weight. We wanted to see if by shifting his diet we could cause the acetone to disappear from his urine; so we increased the diet by 5 gms. of carbohydrates and 10 gms. of fat daily. You will observe that with each successive increase in carbohydrates and each successive increase in fats, it was not until we had his carbohydrates up to 70 gms. with the fats at 180 gms. and the protein at the same level throughout, that the acetone disappeared from his urine. He was a man who came from Pennsylvania. He was anxious to get back to vote; so we did not keep him longer. This, however, will represent to you what you can do yourselves with a mild diabetic. This man's diabetes was of only three years' duration. He was sixty years of age. He came to New York to get iletin, and he went home with merely food. He had something over 2000 calories, and we assured him that he was a very fortunate individual, and that as long as he was carefully supervised he ought to get into no difficulties.

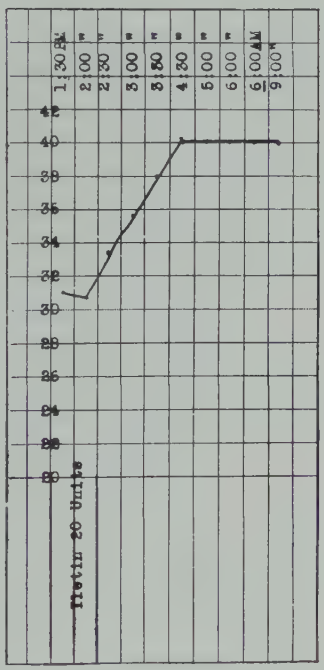


FIG. 70.

Individuals who have acidosis require, for a thorough examination, more than urine studies: for instance, a study of the alkaline reserve of the blood plasma, a study of the expired air and the carbon dioxide content of the blood as an

indication of the existence or the amount of severe acidosis present.

Figure 70 represents a tracing of the alveolar air determination in a girl twenty years old who has a moderate acidosis. She was given 20 units of iletin, and alveolar air determinations were made every half hour. You will see that there was a successive rise in her alveolar air CO_2, that the normal volume was reached in three hours, and that normal was maintained for at least twelve hours. This demonstrates that

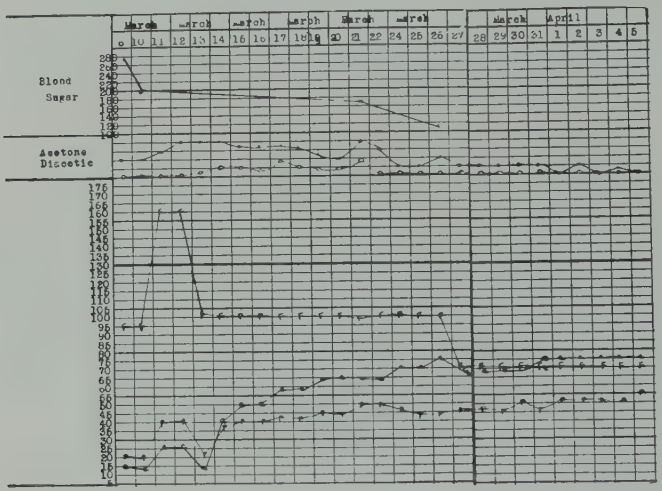


Fig. 71.

what one needs to do to relieve acidosis in diabetes is not to fill the patient with alkali, but to increase the rate of metabolism. It is a matter of velocity rather than amount of metabolism. We know that iletin does increase the fires by causing the combustion of carbohydrate. Instead of pouring in alkali, we give them something which will enable them to burn their acidosis; and in following diabetics this is a method which any doctor can employ.

I would like to call attention to the little alveolar air outfit described by Marriott. Using this apparatus, with care, one can follow grades of acidosis in diabetics sufficiently accurately for clinical purposes. I think I can recommend it to your use.

Figure 71 represents another person who came to us for insulin treatment. She was a woman who had had diabetes for twelve years. I use this graph because it will give you an instance of balancing the diet. On admission her blood sugar was 280 mg. and there was a trace of acetone in her urine, but no diacetic acid. She was placed on a diet of 1000 calories, with 90 gms. of fat. The blood sugar promptly fell. The acetone remained the same. I don't know how we happened to give her such a jump the following day, but we very promptly reduced her fats, for the reason that her acetone curve was rising. You may remember that fats supply 90 per cent of fatty acids, and it is incomplete oxidation of these that brings about ketosis (ketone bodies in the urine). So we promptly lowered her diet to the old level, and then daily increased the diet by 5 gms. of carbohydrates, increasing the protein gradually to a maintenance level of the nitrogen. There was no marked change in her ketone excretion until we lowered her fats definitely, and in fact they did not entirely disappear from the urine until the carbohydrates in grams were higher than the fats. I may say that this individual had no urine sugar from the time she entered the hospital; and this patient would do well not to have insulin. Any diabetic who does not absolutely require insulin will get along more comfortably and better if they do not have it.

Figure 72 is the chart of one who does require insulin. I show this to demonstrate something which I have noted in several of the cases studied. They were for the most part cases of moderately severe diabetics for whom it was necessary to use iletin. This represents the case of a woman who came into the hospital for study. She was placed on Newburgh I Diet, and promptly lost the sugar from her urine. She was feeling poorly, so the diet was increased considerably, and she promptly showed a glycosuria, although at first there was no acetone or diacetic acid in the urine. The diet being kept constant, acetone appeared, and that continued until Jan. 1, when we were asked to give her some insulin. We gave her 15 units three times a day, before each meal, and after iletin was administered she became free from sugar in the urine. The urine was also free from acetone and diace-

tic acid. I have traced out as of particular interest, her fluid intake and output curve. You will see that before she had insulin they had roughly paralleled each other. The broken line represents the output and the solid line the intake. After she had had insulin for a few days we began to see a tremendous difference between her intake and her output. You will observe that where there was an output under 1500 or 1600 c.c. with an intake of something over 3000, she had, as you might suspect, an edema. Not infrequently I have had

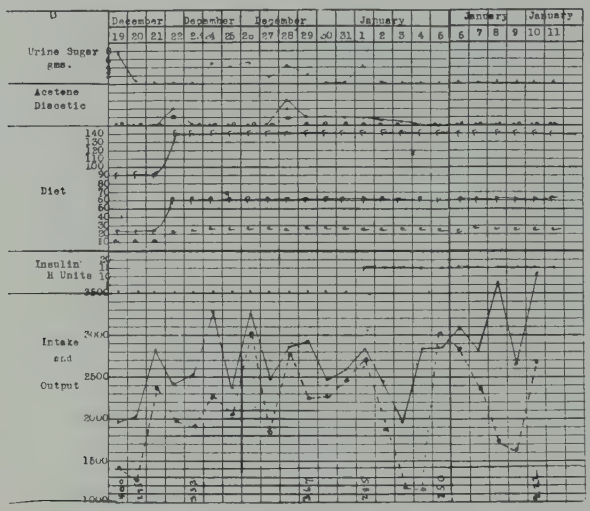


FIG. 72.

patients in the hospital to whom we gave the extract, who went home well improved after two weeks, and who later called up in considerable alarm to tell me their feet and faces were swollen. This shows that fluids may be also concerned in the metabolism of diabetics. The way to control this is simply by salt restriction—sodium chlorid. As soon as I said to those patients: "Discontinue your salt," all the edema disappeared. It suggests a renal impairment of the type we call nephrosis, and the cause is not clear.

Figure 73 is a graph of what one might term acute diabetes. It is the case of a young boy who was in college in Boston and who had, four weeks previous to his admission, an attack of

influenza. He recovered in eight days, and then noticed that he was drinking a great deal of water and passing a great deal of urine. He was examined by a doctor there and sent home with a very gloomy prognosis, and the family felt very anxious. He was brought into the hospital. We put him on a non-nutrient diet for three days and gave him insulin. His diet was raised very rapidly, for he was young and anxious to get back to college. He had done so very well with six days

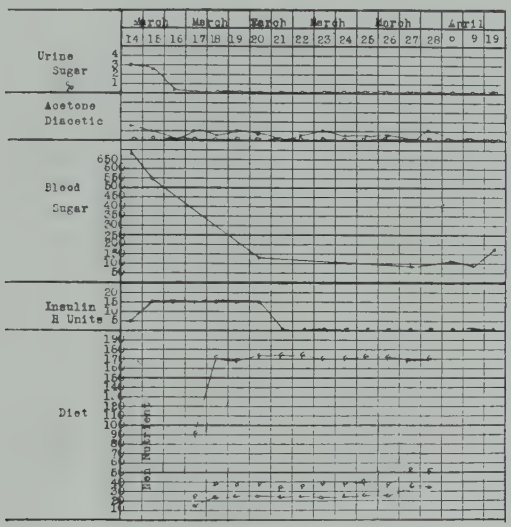


Fig. 73.

of iletin before we stopped it, that we sent him back to school within four weeks. He went through the summer very well because he adhered to his diet. Blood sugar, you will notice, came down to within the normal range, namely, 100 mg., with no urine sugar or acetone. One little rise represents an indiscretion in diet. We found that bread and potatoes should be denied him; but he took things into his own hands this summer, eating liberally of both; and we had him back again this fall for two days of iletin treatment. Then he went back to college and his work. I have wondered all these months whether, in this type of individual, we should add to the

regime small doses of iletin. In the beginning we saturated him with extract and apparently gave his pancreas a complete rest, so that he was able to get back to normal in a remarkable way. He was opposed to insulin injections, and we didn't force the matter. However, I wonder if we should not give these individuals small doses one, two or three times a week to spare the pancreas as much as possible, and thus attempt complete cure.

Figure 74 is the graph of a man who had nephritis and diabetes. He came in with a marked acidosis. In fact, his

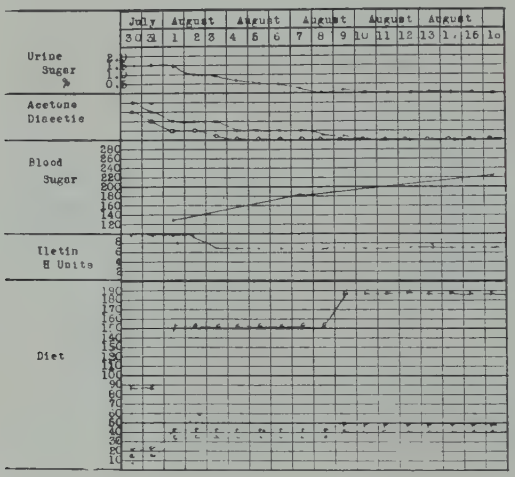


FIG. 74.

doctor sent him, thinking he was going to die, and wanting to see what we could do for him. His blood-sugar figures were not taken on that day, unfortunately. This study was made during the period when I was trying to simplify things. We promptly began the use of iletin and gave him 2½ units every six hours. He was the type of individual that would be expected to go into coma, except for his edema. From the very first his general condition improved, even with small doses of iletin, and so after a day or two of low diet (he was on 1000 calories—starving) we raised his diet, his blood sugar rose; we lowered his extract to three doses a day of 2½ units, he promptly lost his glycosuria; gradually he lost

his ketone bodies and he went home with a blood sugar of over 200, with no sugar in his urine, and happy in the thought of being able to go back to work in a few weeks. He runs from 1 to 4 gms. of albumin per liter per day, and he has to be upon a salt-free diet. His general condition is not good. These individuals do not warrant a very rosy prognosis. The first one I had experience with was a patient with nephrosis in an extreme stage. He was completely edematous, and every time I gave him a dose of insulin and decreased his blood sugar, he felt so bad and was so dispirited that I abandoned absolutely the treatment of his diabetes

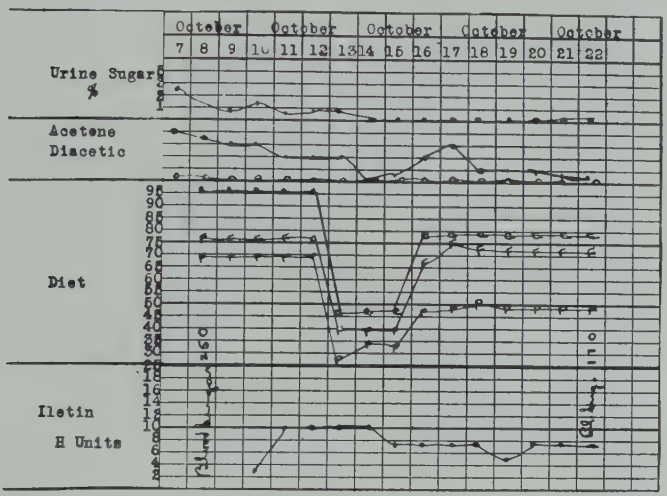


FIG. 75.

and tried to do what I could for his nephrosis. We did not have a great amount of success with him.

These patients were of the type we call nephrosis. Such a condition represents a serious complication; and in making a prognosis this should be taken into consideration.

Diabetes in Children. Figure 75 is a graph of a little girl who had diabetes of six weeks' known duration. She was ten years old, and she was sent into the hospital to be regulated and studied. We placed her first on a milk diet, 6 oz. every three hours, night and day, which would give her the proper amount of nitrogen and fluid and a sufficient

amount of calories. Iletin was given her for the first few days. Then we replaced the grams of carbohydrate, fat and protein in milk by other food and began giving her small doses of extract to see what she could do with it. She had only two blood-sugar determinations. She had 250 mg. soon after admission. We began dropping the diet a little, shifting it around, and placed her upon the following diet and she gained weight: protein, 50 gm., carbohydrate, 28 gm., fat, 65 gm. We sent her out with a blood sugar of 110 mg. and

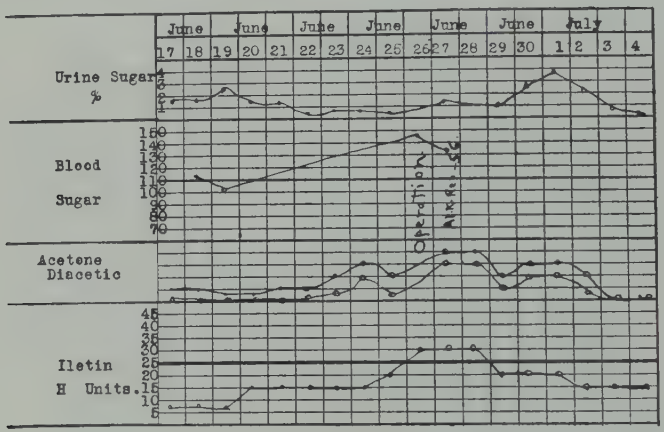


Fig. 76.

7½ units of iletin; and she should have it continuously. That amount will never hurt her. I have yet to give any patient small doses of 2½ units of iletin and see any undesirable effects, except in very thin debilitated individuals.

Diabetes in Surgery. Figure 76 is the graph of a woman who came in for operation. She had a huge ovarian cyst with a large umbilical hernia. She came in for a period of study, being operated upon on the 26th of June. She was a non-cooperative diabetic, for she was kept supplied by her family with candy. We tried to put her on a diet, but could do little for her. She starved herself in certain ways, and had a very definite ketosis with which she went into operation. Immediately after, during the time of operation and while on the table, saline was given, She was given 1500 c.c. of

saline under the breast while on the table, and every twelve hours 500 to 1000 c.c. of saline were given under the breast. Her alkaline reserve, which would be an index of her acidosis, on the day following operation, was 56. So she was in no danger of acidosis, although she certainly had a great number of ketone bodies. She went home and made a perfectly uneventful recovery. Complete healing of the wound took place. During all this period she had a glycosuria. It is fair to assume she had a considerable hyperglycemia, but this made no difference in healing of her wound.

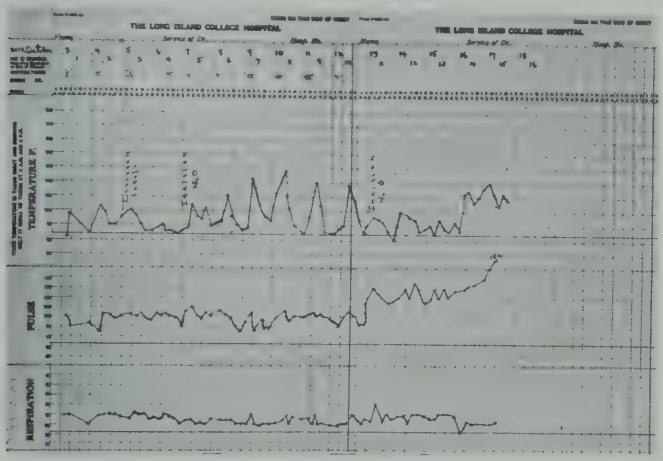


FIG. 77 *A*.

The healing of the tissues has been especially interesting to me in all the surgical cases. We have looked upon hyperglycemia as one of the reasons why wounds do not heal. Here was primary repair in the presence of a frank hyperglycemia.

Figures 77 *A* and *B* represent our first experiment in a serious case of diabetes and infection. This woman had a tremendous infection of the right hand. The whole hand was dead. The bones were easily seen and she had pus all down the tendon sheaths. At the time she came in she was in a extreme acidosis. Her alkaline reserve was under 20, her blood sugar was high and her skin and mouth were dry. Her tongue was a fiery red. She said: "I don't know what is

the matter with me, I feel like a dope." A typical picture of acidosis of severe type. Large amounts of extract were given her and she was fed with carbohydrates all the time. The urine sugar was always high. She always had acetone and the diacetic acid disappeared, but returned. She was getting 40 units of iletin a day and there was a period when she would have been a good surgical risk. I think had we taken off her hand at that point she would now be here instead of

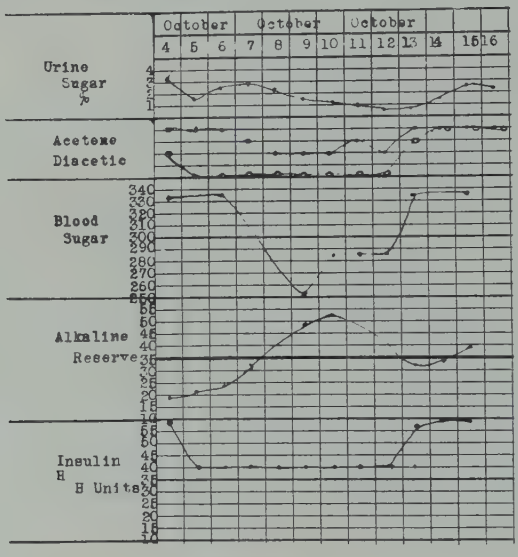


FIG. 77 *B*.

being no longer with us. There were, however, family reasons why we could not go ahead and operate, and the patient died.

Having learned that lesson, when, sometime afterward, they brought me in an old lady with an equally serious infection of the foot, I made up my mind we would clean up the infection; so within three days we had her leg off and had her out of the danger zone. She made a complete recovery and is now walking around on an artificial leg, and is very thankful she came to the Long Island College Hospital (Fig. 78 *A* and *B*).

Figure 79 is the first chart of a boy, a non-cooperative diabetic, who came into the ward. We never could get him

to do anything right. He had severe acidosis. We gave him large doses of insulin and carbohydrates during all this period. He had any amount that he wanted of cereal, bread

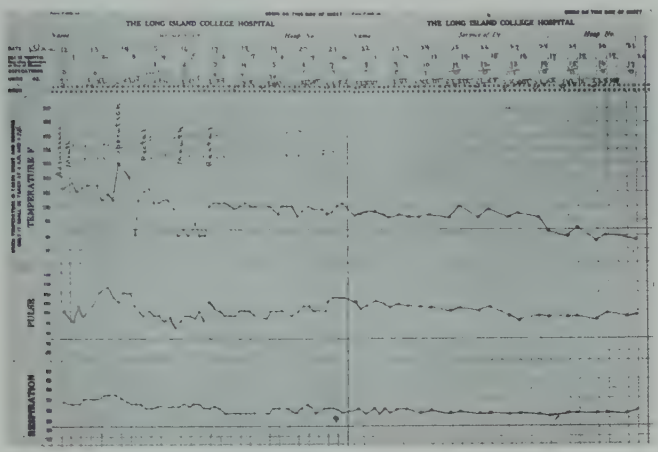


FIG. 78 *A*.

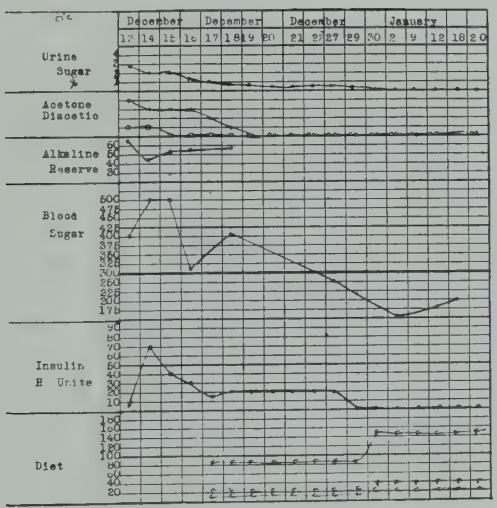


FIG. 78 *B*.

and cream. We relieved his acidosis. He was no longer sick, but felt very well, and was still getting comparatively large amounts of iletin. His diet was restricted to a low level,

but his urine sugar showed a marked rise and we discovered that he was preparing nice little foods for himself at night.

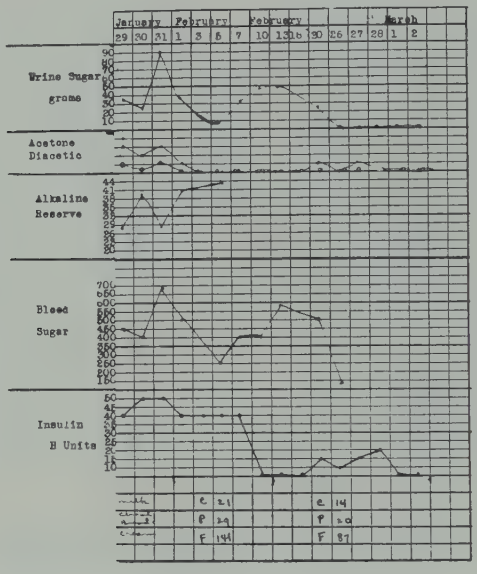


Fig. 79.

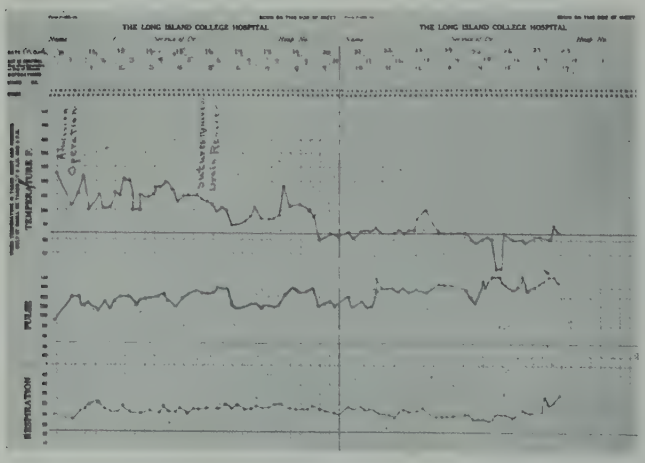


Fig. 80.

Consequently we put him on a starvation diet and kept him in bed, and we finally sent him home without any

sugar in the urine, with no acetone bodies, and with the blood sugar within normal limits. He came back in seven days with an acute appendix and was operated upon. He had a peritonitis. Figure 80 represents his bedside chart and will give you an idea of his clinical course. Figure 81 is the graph covering this period. He had considerable acidosis immediately after his operation, but we were able to relieve that as soon as we dieted him. There was a considerable fall in the blood sugar. We gave him always large

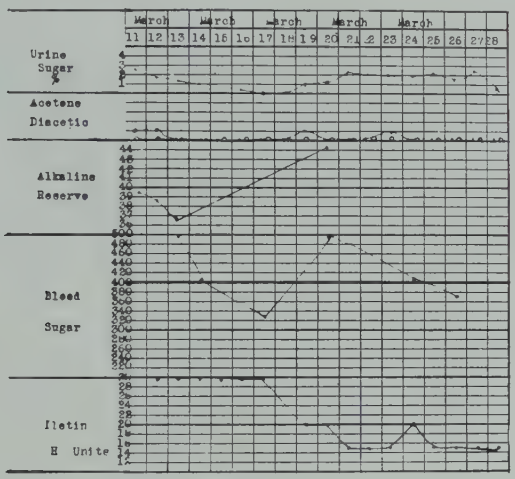


FIG. 81.

amounts of iletin, although later we decreased that. At this point he was taken home by his parents and he died a few days later.

The point I want to make is that acidosis, even in untreated diabetics, is a thing we do not need to fear in surgery. But in a severe conflict the non-cooperative diabetic handicaps himself and falls.

I want to contrast the case just mentioned with that of Figures 82 and 83, a boy twenty years old. He had severe diabetes with acidosis. He came into the medical ward for regulation of his diabetes which had been of sixteen months' duration. He began to have abdominal pain, and it was very apparent that he had an acute appendix. He was

operated upon on the 7th of the month. During all the period immediately after operation he had a considerable hyperglycemia. His acidosis was not marked. We were finally able to discharge him upon a diet which enables him

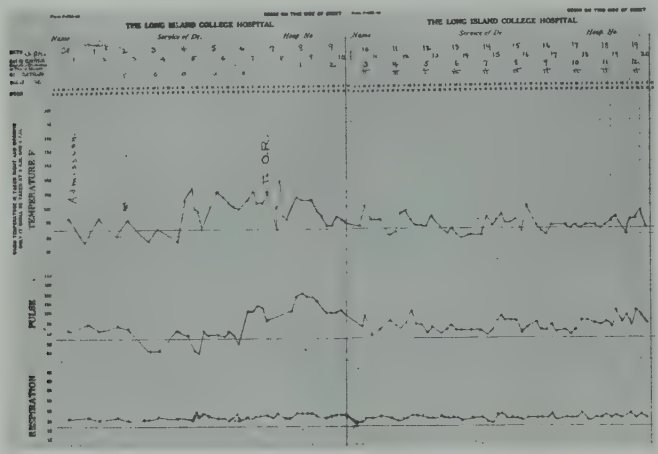


FIG. 82.

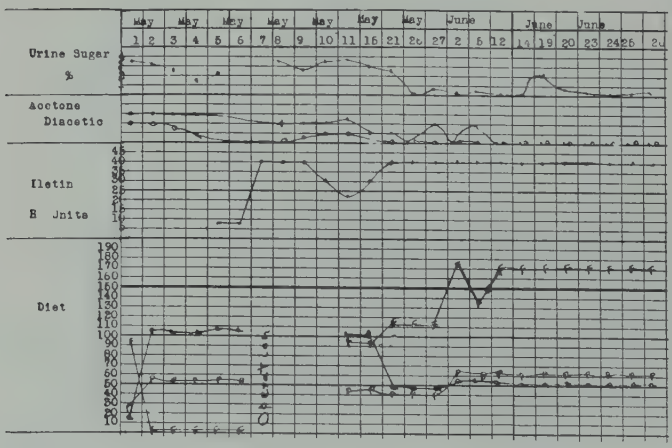


FIG. 83.

to carry on his work (which was that of driving a truck) although he is still taking 40 units of iletin daily. Cooperation on the part of the patient is still necessary for successful treatment.

Figures 84 *A* and *B* represent the case of a woman who had an infection in her foot. She was sent to me from New Jersey because of alarm about her condition. She represents

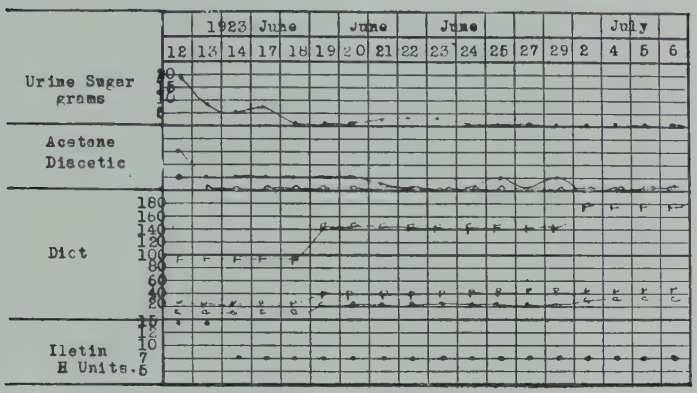


Fig. 84 *A*.

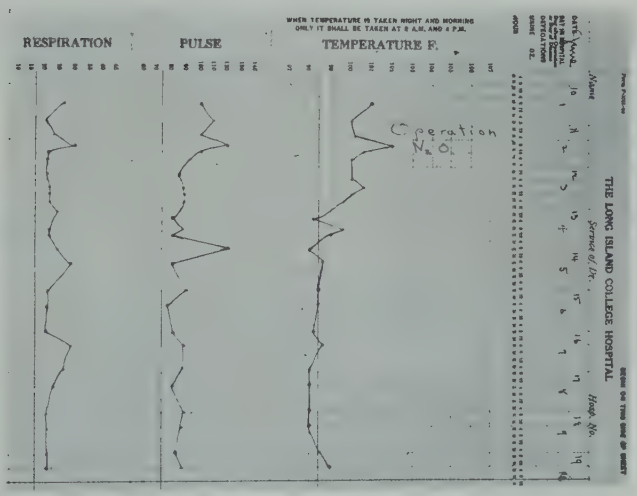


Fig. 84 *B*.

the type that can be managed without going to the hospital; that is to say, a doctor doing minor surgery can take care of such a case in her home, provided he gives her small amounts of iletin—repeated small doses. She had 6 doses a day on

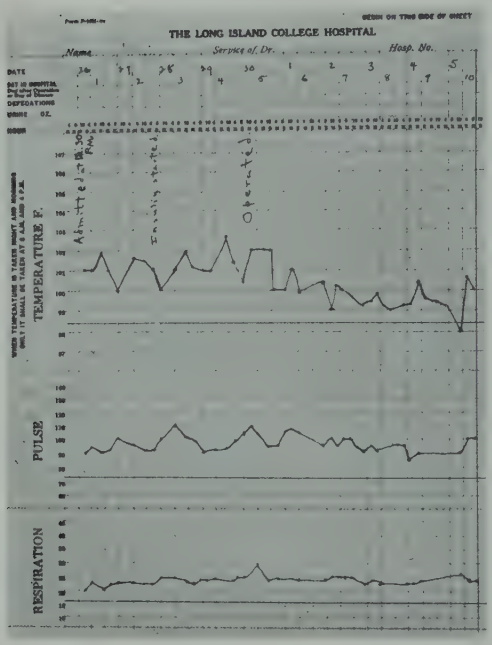

THE LONG ISLAND COLLEGE HOSPITAL
Name
Service of Dr.
Hosp. No.
DATE
TEMPERATURE F.
PULSE
RESPIRATION

FIG. 85.

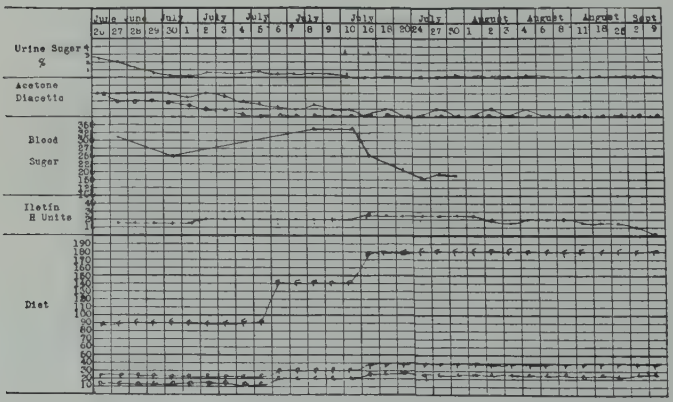

June June July July July July July July August August August Sept
Urine Sugar %
Acetone Diacetic
Blood Suger
Iletin U Units
Diet

FIG. 86.

her first admission and then went along, getting 7½ units a day. Her diet at first was simply a starvation diet and then a maintenance one. She promptly lost the sugar from her urine and also her acetone and diacetic acid.

Figures 85 and 86 represent the case of a woman who had a tremendous infection in the muscles of her thigh. She came in in extreme acidosis; and she was one in whom I trusted practically to purely clinical determination, such as any practitioner knows. You will see a blood-sugar curve, merely as a matter of interest. She had had a severe acidosis for a long time, and had been sick for two weeks at home. When she came into the hospital this abscess in the muscles of the thigh was not pointed, and it was not until four days after admission that she was operated upon, because the abscess had not localized, and not because we were afraid of any acidosis. The point I wish to make is that even though she had acidosis, even though she had a severe infection, she was cared for with small doses of iletin. She now gets no iletin. She is perfectly sugar-free and is up and about.

To sum up, I would affirm that all diabetics do not require iletin; also that no man should attempt to care for a diabetic unless he is willing to give the diet careful consideration and is willing to see that the patient does not get into trouble. Otherwise, he ought to be willing to let somebody else take the case. On the other hand, the majority of diabetics are mild cases. They must fall to the care of all of us; and I think now that the only individuals I would send to a hospital are those who are in coma, or who have severe infections. I would not undertake to care for any case of coma at home. The needs are too numerous and too complicated, and time is a very telling factor. In severe acidosis, in cases where there is discomfort from itching, I believe in the use of large doses of iletin—rapid de-sugarization with careful attention. In all other cases, small doses can be given. Give 6 or 8 doses a day of 2½ units each, and your patient will steadily and slowly improve. To those of you who are opposed to the use of iletin, I would suggest that you experiment very carefully with it and then decide whether to use it, as a routine.

SUBACUTE BACTERIAL ENDOCARDITIS IN THE ACTIVE AND HEALING STAGES*

EMANUEL LIBMAN, M.D.

THE main object of this paper is to present some of the features of the disease now called subacute bacterial endocarditis, and to attempt to make clear what is meant by the bacteria-free stage of the disease. This is not as well understood as it should be.

Before discussing the question of endocarditis, it is important to take up and emphasize the difference between valvular defects and endocarditis—a valvular defect being a finished process, and an endocarditis being an active process, which may or may not be superimposed on a previous valvular defect.

The causes of valvular defects are as follows:

Rheumatic fever is the most common. Next in frequency is *syphilis*. *Atherosclerosis* causes a great many valvular defects, but they do not present the same appearance as the defects produced by rheumatic fever. In the aortic valve there is produced stiffening, obstruction and occasionally, insufficiency: in the mitral valve one finds thickening of the valve and at times calcification at the ring. Congenital lesions also cause valvular defects, as does, rarely, trauma. A valvular defect can also be produced by an *acute bacterial infection of a valve*, which, after causing destruction of part of the valve, heals. This is a very rare occurrence. It has been suggested by some, particularly by Sir Thomas Lewis, of London, that valvular lesions might be produced by recurrent attacks of *subacute bacterial endocarditis*. A certain amount of evidence has accumulated which makes it appear that this disease, particularly in recurrent form, can produce at least marked thickening of the valves.

* This lecture was delivered on April 6, 1923.

It is essential to define what is meant by rheumatic fever. There is a great need of definiteness in talking on this subject. By rheumatic fever we mean a disease which is characterized clinically by involvement of the joints, with swelling, pain, perhaps redness, and by a tendency for the inflammation to travel from one joint to another. Suppuration never occurs in this condition. In association with this inflammation of the joints, or preceding it, or following it, there is

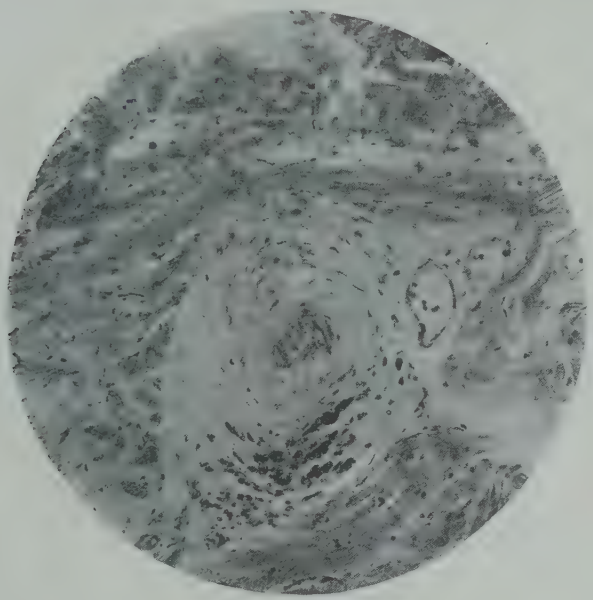


FIG. 87. Aschoff body, situated in the adventitia of a small artery. Stained with methyl-green pyronin. (After Thalhimer and Rothschild. *J. Exper. M.*, 1914, xix.)

apt to be developed endocarditis, tonsillitis, pericarditis and certain skin lesions, one of the most characteristic of which is erythema annulare or gyratum.

In *rheumatic endocarditis* there is a secondary invasion of the blood-current by anhemolytic streptococci in at least 8.3 per cent of the cases. This sometimes causes difficulty in distinguishing these cases from cases of subacute streptococcus endocarditis. There are differences in the streptococci found in these two conditions, but it requires careful study to

differentiate them. In rheumatic fever there are no immunological reactions against these cocci, whereas in subacute streptococcus endocarditis the serum of the patient always gives a positive complement fixation reaction against the particular streptococcus found in the case.

It is important to remember that in rheumatic fever, we do not know where the primary seat of the virus is. It is believed that the tonsils may be the primary focus, but it is just as possible that this may be only one manifestation of

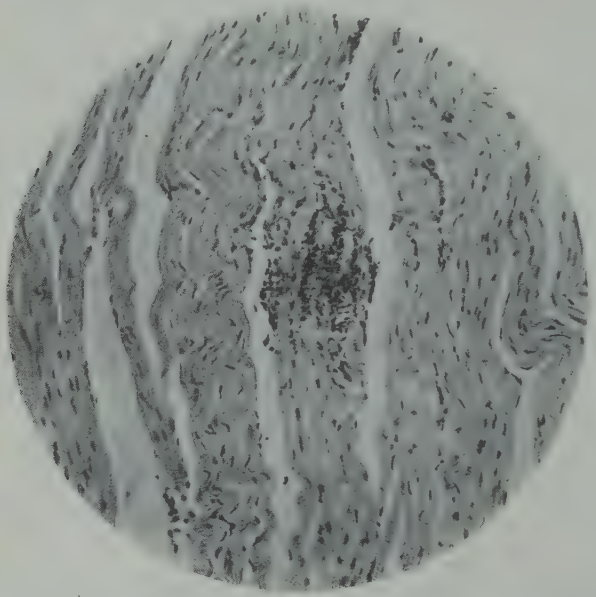


Fig. 88. Bracht-Waechter lesion. Earliest stage of focalized myocardial lesion with beginning degeneration of muscle fibers. Hematoxylin and eosin. (After Thalhimer and Rothschild. *J. Exper. M.*, 1914, xix.)

the rheumatic fever, because removal of the tonsils may prevent attacks of rheumatic fever, but does not prevent the development of chorea.

The only pathognomonic lesion which we know of in rheumatic fever is the Aschoff body, which occurs mainly in the myocardium, sometimes under the endocardium and sometimes in the pericardium. It is a perivascular lesion which is characterized in the main by very remarkable large cells with one or more large polymorphous nuclei,

the cells being arranged in the form of a fan or rosette. These large cells stain in a particular way with certain dyes.

In cases of subacute bacterial endocarditis there is often present a focalized lesion in the myocardium, known as the Brach and Waechter lesions. These are foci which consist

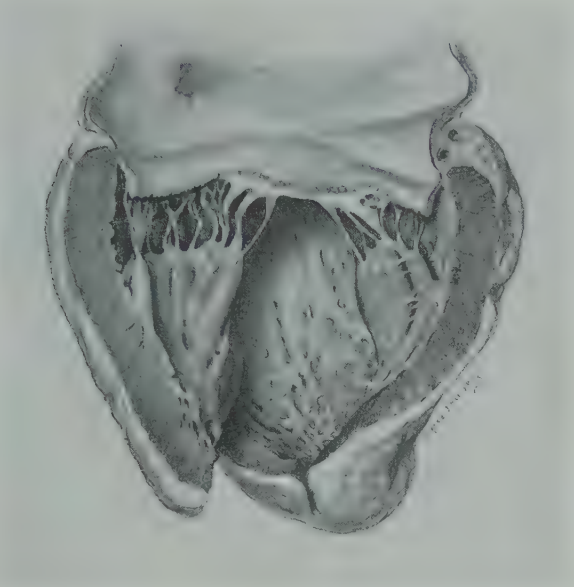


FIG. 89. Rheumatic endocarditis. There is a practically continuous row of minute verrucous vegetations typical of rheumatic endocarditis. On the aortic cusp the vegetations are situated on the closure line, whereas on the posterior cusp they are situated on the free edge which now coincides with the line of closure. There is a fan-shaped thickening of the valvular attachments of the chordae tendineae of the aortic flap. Aschoff bodies were present in the heart muscle and there were necroses in the heart muscle and the cortex of the kidneys due to a vascular lesion.

mainly of lymphocytes and are found in the muscle fibers themselves—the Aschoff bodies being found outside the muscle fibers. The Brach-Waechter bodies have been reproduced experimentally, but nobody has been able to reproduce the Aschoff bodies.

Before discussing the clinical and pathological features of subacute bacterial endocarditis, it is necessary to discuss

briefly the various types of endocarditis which are encountered.

The most frequent form is rheumatic endocarditis, which occurs in acute, subacute and recurrent form, and probably not in any permanently continuous form. As is well known, it attacks healthy valves with the greatest frequency. The lesion is always verrucous, a number of small separate verrucae being developed from an independent base. Syphilitic endocarditis may possibly occur in acute and subacute

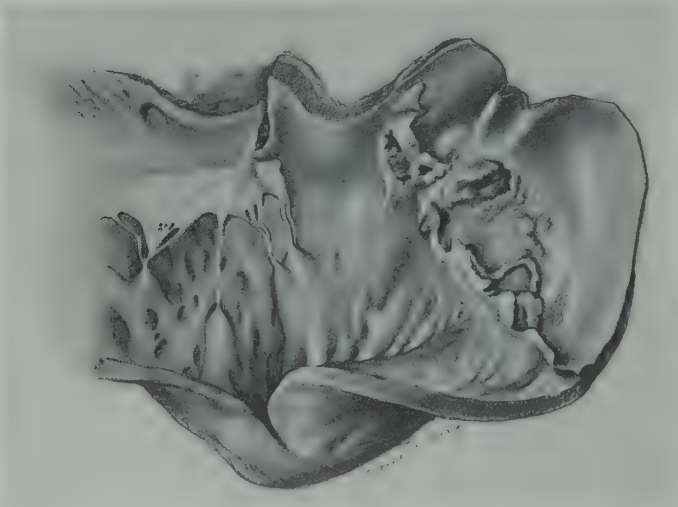


FIG. 90. Acute bacterial endocarditis. Sinococcus infection. Almost complete destruction of pulmonary valve.

forms, but practically all we know about it is the end-result in the form of aortic insufficiency. The third group comprises cases which are due to known bacteria—and which I have called the bacterial group. The question might be asked why I do not include rheumatic fever in this group. There is no doubt but that it is due to some kind of microorganism. But this has not yet been found, so that it is practical to call those cases which accompany rheumatic fever, rheumatic endocarditis and those cases which are caused by known bacteria, bacterial endocarditis. When the inciting agent of

rheumatic fever is found we can introduce a proper nomenclature.

The bacterial cases occur in two forms, one of which, the acute, is much less common than the other, the subacute. For many years chief attention was paid to the former, which was called "acute malignant endocarditis."

By acute cases we mean those running a course of about six weeks. These cases are caused by hemolytic streptococci, staphylococci, pneumococci, meningococci, the gonococcus and other gram-negative organisms. A great variety of other organisms can initiate the disease. Occasionally an acute case can be attributed to an anhemolytic streptococcus or the influenza bacillus. The subacute cases are due in 95 per cent of instances to an anhemolytic streptococcus, the so-called streptococcus viridans. The name streptococcus viridans is not appropriate, because many of these cocci do not grow in the form of green colonies. The remainder of the cases not due to streptococci are nearly all due to Pfeiffer's bacillus, the so-called Bacillus influenzae. It is important to keep in mind the fact that this does not indicate any relationship to the disease, influenza. In the subacute cases there may be a secondary invasion of the blood by a typical pneumococcus. That occurs particularly in cases in which an intercurrent pneumonia develops.

Besides the rare cases of tuberculous endocarditis and the valvular inflammations that occur in the course of the specific infectious diseases and the exanthemata, there is a large group of verrucous endocarditides designated as terminal or cachectic because they are found to be present at post-mortem examinations made in cases of chronic diseases, such as nephritis, diabetes, carcinoma, etc. The condition is found quite frequently in cases of leucemia. The lesions consist of small verrucae, and occur mainly on the mitral valve. It is believed by many that the condition is toxic in origin, but it is much more likely that it represents some form of infection. There are no characteristic lesions in the heart muscle.

Furthermore, there are cases of verrucous endocarditis, which are apparently not rheumatic in origin, and which are definitely not terminal in character. This is a class of cases

which we believe it is best to designate for the present as "atypical verrucous endocarditis." This group does not correspond to subacute or acute bacterial endocarditis, because bacteria are not demonstrable by ordinary methods. It does not correspond to rheumatic endocarditis, because both the gross and the microscopic anatomy are quite different. There are no characteristic lesions in the heart muscle, such as occur in rheumatic endocarditis. There is a tendency in these cases to the development of mural endocarditis, which is not often found in rheumatic cases. It is particularly important to look for this lesion in cases of severe purpura. A peculiar feature of these cases is the involvement of the cusps of the pulmonary valve—this valve not being involved in rheumatic endocarditis and practically never in subacute bacterial endocarditis.

We now come to the type which I wish to discuss today somewhat in detail, namely, the cases known as subacute bacterial endocarditis. This disease was formerly known as chronic malignant endocarditis, chronic infectious endocarditis— and by some is called endocarditis lenta. As the disease is almost entirely due to streptococci, it is permissible to designate it as subacute streptococcus endocarditis. In any case in which the influenza bacillus happens to be found, one may use the term subacute bacillus influenza endocarditis. This disease is much more common than was formerly believed to be the case. Dr. Edward G. Janeway, as far back as 1899, reported that he was seeing this disease quite frequently. Our studies are based on an experience covering over 500 cases.

The cocci found in this disease are called anhemolytic, because clear areas do not develop around the colonies on the plates made up of agar and blood. There are transitional forms between these cocci and typical hemolytic streptococci. Experimentally, endocarditis has been produced with anhemolytic streptococci obtained from cases of endocarditis, but exactly the same lesions have been found in studies made with saprophytic anhemolytic streptococci found in the feces.

The organisms are found in the blood in these cases with varying frequency by different observers. In 1910, Dr. Celler and I reported 73 cases, in 69 of which cocci were found, and

in the other 4, the influenza bacillus. This is a much higher percentage of positive results than has been obtained by most investigators. It is important to remember that there are cases in which the blood cultures are negative throughout the course of the disease, and yet at post-mortem examination the vegetations in the heart will be found full of cocci. By bacteria-free cases we mean cases in which at autopsy few or no bacteria are found. Whether the cocci are found during life or not, does not mean anything as regards the classification into bacterial or bacteria-free.

The foci from which the streptococci enter the blood are believed to be, in the main, the tonsils, infections around the teeth, the accessory sinuses, the bronchi, and, possibly, the intestinal tract. There is a great probability in certain cases of pneumonia that the pneumococcus enters the blood, is deposited in a valve and is changed over to an anhemolytic streptococcus, which later causes the development of an endocarditis. These streptococci occur in the blood in conditions other than endocarditis. In a variety of infections these organisms have been found in the blood, and at times the organism may simply represent a transitory invader in the course of various diseases. When the organism enters the blood from an infected uterus, a differential diagnosis must be made between that condition and a subacute streptococcus endocarditis that antedated labor, but was not recognized.

We will now pass to a consideration of the clinical picture of the disease. The onset is usually insidious. There are great differences in the way in which the disease begins in different cases, but, most commonly, the patient complains first of lassitude, pains in the legs, loss of appetite, feverishness, chilliness, headache and occasionally cardiac symptoms. In certain cases the onset apparently is very acute. The constant symptom is the fever. Other symptoms that may occur are chills, sweating, emaciation, leucocytosis, petechiae, purpura, embolisms of various kinds, embolic aneurysms, tender cutaneous nodes, cardiac symptoms, tenderness of the sternum, enlargement of the spleen, gastrointestinal symptoms, cerebral symptoms, swelling of the joints, renal symptoms, various pains, and changes in the eyes. The

clinical picture is a very protean one. It will not be possible today to discuss in full the clinical phenomena in this disease.

In general, the symptoms may be classified into three groups: First, there are those symptoms which are due to toxemia and the anemia resulting from it. Second, there are symptoms which are due to pieces breaking off from the valve—these we call endocarditic symptoms. Third, there may be symptoms from myocardial weakness. It is important to remember that the cardiac symptoms are very often not marked in cases of this disease. In a large number of the cases in which cardiac symptoms develop, they are due to weakness of the heart muscle from anemia and toxemia, rather than valvular defects as such.

The fever is of various types. It may be continuous at times, and at other times it is very irregular. There are cases in which the elevation of temperature is very slight, and for a week or two before death, especially in cases of renal insufficiency, fever may be absent.

In the lungs there may be found bronchitis, bronchopneumonia, pleurisy and hemorrhagic infarctions. The phenomena due to involvement of the kidneys are among the most interesting. Three main types of lesions may be found. In the first place, there is an embolic glomerular lesion which causes damage only to parts of a certain number of the glomeruli. Hematuria, either gross or microscopic, may result, but the leson is of no importance as far as danger to the life of the patient is concerned. The diffuse glomerular nephritis which may occur in this disease is of much greater importance. It is interesting and important to note that cases which come under observation in the bacteria-free stage present glomerular nephritis at least fifteen times as often as it occurs in the active stage of the disease. Some observers believe that glomerular nephritis occurs when the bacteria-free stage develops, because of the killing off of the bacteria. The third type of lesion which occurs is embolic in nature and is characterized by the presence of small or large infarcts. In many cases remarkable symptoms are produced by these lesions. Besides hematuria and albuminuria, there may be tenesmus, frequency of urination, pains in the back referred to the chest (even as high as the

shoulder). These symptoms often lead to an erroneous diagnosis of tuberculosis of the kidney or pyelonephritis.

The spleen is often enlarged in this disease, the enlargement sometimes amounting to a splenomegaly. Perisplenitis may occur, due to infarction. The pains in this condition may be situated over the spleen, or may be referred to the anterior part of the chest or to the shoulder. The gastrointestinal symptoms are varied. There may be distress after eating, anorexia, vomiting, diarrhea and occasionally, jaundice. Pain in the region of the stomach or in other parts of the abdomen is very frequent. When the gastric symptoms predominate, an erroneous diagnosis of carcinoma of the stomach may be made.

The blood examinations give, in a way, very interesting results. There may be a high leucocyte count with a marked increase in the polymorphonuclear leucocytes. On the other hand, the count may be normal or subnormal, and with this there may or may not be a lymphocytosis. The hemoglobin index is never over 1, unless nitrogen retention is present. The presence of histiocytes (macrophages) is important in the diagnosis.

Of great importance for the diagnosis are some of the cutaneous phenomena. The most frequent lesion found in the skin is petechial in nature. The petechiae with white centers are the most important for diagnosis. They should be looked for particularly in the conjunctivae, the sides of the neck and the fundus of the eye. The center of the petechiae is not elevated. Petechiae with elevated centers practically always mean a general staphylococcus infection with or without endocarditis. Purpura is not common in the active stage of the disease. It may be embolic in origin or due to changes in the blood, or in the walls of the vessels.

Less common, but even more important, for diagnosis, are the tender cutaneous nodes which are now generally called Osler nodes. These are most commonly found in the pads of the fingers and toes, the thenar and hypothenar eminences, the sides of the fingers and the web between the toes. They appear at intervals as small swollen areas, some of the size of a pea and others as large as 1½ cm. in diameter. They may disappear in a few hours, but usually last a day or longer.

Sometimes they are only pin-point in size. No matter how small they are, tenderness is always present. The lesions are usually pinkish in color, but may appear bluish or purplish. At times one finds only pale, firm areas in the skin; such areas are also tender. The tender node of Osler is the one pathognomonic symptom of the disease. It should be distinguished from the lesion which we call the Janeway lesion, which occurs in acute bacterial endocarditis. This lesion consists of small erythematous or partly hemorrhagic lesions, which may be slightly elevated, and which are found in the soles of the feet and the palms of the hands. They are not tender and do not have the real nodular character of the Osler lesions. They are found rarely in the subacute type of the disease.

Emboli may occur in vessels in different parts of the body. Embolic aneurysms may be produced by destruction of the wall of a vessel in which an embolus is located. These embolic aneurysms cause pain, and may, when ruptured, cause hemorrhages.

In a number of cases, as the disease progresses, a peculiar café-au-lait pigmentation of the face may develop. Rarely a peculiar erythematous lesion may be found in the nose and adjacent parts of the face.[1] The color of the face does not become deep brown except in the bacteria-free stage.

Sternal tenderness is a symptom which occurs in some of the cases, particularly late in the disease. It is elicited by tapping the upper and lower part of the sternum and comparing the sensitiveness in these two regions. This symptom is found more often and to a more marked degree in the bacteria-free stage.

A great variety of cerebral symptoms may occur. A particularly interesting condition which has been found in a number of cases is a polynuclear meningitis, in which streptococci may or may not be found. Besides symptoms due to plugging of various vessels, and symptoms due to toxemia, there may be found a subarachnoid or intraventricular hemorrhage, the two latter conditions usually being brought about by rupture of embolic aneurysms. Paralyses of the eye muscles are uncommon.

[1] This has been observed only twice in patients who were treated out of doors.

While joint swellings may be found in this disease, they are never acute, and redness of the skin over the joints is not encountered. In this respect the disease differs from rheumatic fever. Pericarditis is not a part of this disease. It might occur due to an intercurrent pneumonia or to renal insufficiency.

Besides petechiae which may be found in the fundus of the eye, optic neuritis, and even choked disk, is not rare. According to Falconer, of London, optic neuritis is found in one-third of the cases. For the purposes of diagnosis, therefore, it is important to make an ophthalmoscopic examination in these cases.

Subacute bacterial endocarditis occurs particularly in patients who have suffered little or not at all from their cardiac disease. Some of the patients who develop this condition do not know that they have had a valvular lesion. Others have been informed that such a condition was present in the course of examination for insurance, or for some other purpose. Patients who have fibrillation of the auricle rarely develop this disease, and it is uncommon for a patient who has suffered from a subacute bacterial endocarditis to develop fibrillation.

The disease usually lasts four to eighteen months, but some cases run a course of two years or more. The most common causes of death are exhaustion, cardiac insufficiency and complicating conditions, especially pneumonia. In a few cases the termination occurs because of uremia, but this mode of ending of the disease is much more characteristic of cases in the bacteria-free stage.

In the last twelve years we have learned much that is new concerning the course and outcome of cases of subacute bacterial endocarditis. In the discussion of this subject it is necessary to distinguish cases in the bacterial and bacteria-free stages. The case is surely in the bacterial stage if the blood culture is positive; but it may be in the bacterial stage, and yet the blood culture may be repeatedly found negative. This can be determined by clinical criteria, but more definitely by the post-mortem examination. In cases in the active stage of subacute bacterial endocarditis, the surface of the vegetations consists almost entirely of bacteria. If the

organisms are present in small numbers, the case is already going over to the bacteria-free stage. With these facts in mind, we can discuss the cases as they present themselves to us clinically.

I. CASES GIVING A POSITIVE BLOOD CULTURE

More than 90 per cent of cases in the active stage are found, if proper methods are used, to have bacteria in the blood. What happens to these patients? Nearly all of them die. When I had completed the study of the first 150 cases, I had observed 4 cases of complete recovery. In these cases there was no proof that the particular method of therapy employed was responsible for the favorable outcome. Since that time I have observed at least 2 more such cases. The first 4 patients came under observation, respectively, nine, seven, five and five years ago. The fifth patient was observed during the greater part of the year 1921, and has been free from the infection since January of this year. The sixth patient that recovered has been under observation for several months since the time of recovery. To be sure that a patient has really had the disease, one needs more, as I stated above, than to find that there is present valvular disease and that there are bacteria in the blood. There must be definite clinical symptoms present, such as fever, splenic enlargement, petechiae, tender nodes, embolisms and progressive anemia. There exist cases in which a positive blood culture is found followed by negative blood cultures. These patients usually die within a few months. After the blood is free from bacteria, the patients generally present symptoms like those occurring in the cases to be described directly, that come under observation for the first time in the so-called bacteria-free stage.

II. CASES COMING UNDER OBSERVATION WITH NEGATIVE BLOOD CULTURES

A. Some of these cases are in the active stage. Clinically, they act like the cases in which the blood cultures are positive, and the necropsy shows vegetations full of bacteria. These cases really belong in Group 1, constituting cases of that type in which we failed to find the bacteria during life.

B. This group has the same symptoms as cases in the active stage, but they gradually lose the fever and most of the other symptoms. Most of these patients die while still in the febrile stage. At the necropsy, few or no bacteria are found in the vegetations. They are evidently cases that were recently in the bacterial stage of the disease. Those patients that live after the fever has disappeared, present a clinical picture like that presented by patients constituting the next group.

C. Here we find a remarkable group of cases in which fever is not a characteristic symptom, as it is in the active stage of the disease. Some of these patients have no elevation of temperature during a long period of observation, which may extend over many months. Fever may occur because of complications, anemia, or intercurrent diseases. The type of cases belonging to this group, which comes under observation still febrile, with negative blood cultures, and then becomes afebrile, has been described in Group B.

In papers published in 1912 and 1913 and in subsequent communications, I have directed attention to a group of patients who come under observation with a valvular defect and with a variety of symptoms, such as embolisms of the various types mentioned above, progressive anemia, splenomegaly, progressive insufficiency of the kidney due to subacute or chronic glomerular nephritis, or a peculiar pigmentation of the face. These cases had generally been considered to be instances of chronic rheumatic endocarditis. From pathologic, bacteriologic and clinical studies, it was definitely shown that such patients must have had a previous endocarditis of the bacterial type, and had not come under observation in the bacterial stage, or had not been recognized as suffering from such a condition. Such cases are designated as cases of subacute bacterial endocarditis that have become spontaneously bacteria-free. The lesions in the heart were of the same type as were found in the cases in which positive blood cultures had been obtained, but showed marked or complete organization with or without calcification. In nearly all the cases the typical embolic glomerular lesions were found, but in a healed stage. They were much less abundantly present than in the cases that

showed evidence of active infection. In the few cases in which the embolic glomerular lesions were not found, the patient had presented typical Osler nodes during life, and the cases could, therefore, be accurately identified.

It is interesting to speculate on the probable great frequency of subacute bacterial infections of the valves of the heart with complete recovery in cases in which there is no history of the active infection. I have shown that cases with positive blood cultures may undergo complete recovery, and have drawn attention to the occurrence of cases coming to us with sequelae of the disease in which there is no history of the active bacterial stage of the disease (we have seen at least 50 such cases). It is, therefore, most probable that there occur cases in which the infectious stage was never recognized clinically, and in which a complete recovery ensued, leaving no evidence behind of a type that we have heretofore recognized.

III

Recurrences. During the last year we have made the novel observation that recurrences of the disease exist.

We will now discuss briefly the pathology of the disease and then the group of cases under heading C which came under observation after they had become spontaneously free of bacteria.

We will describe the lesions with the greatest brevity. The important changes in the lungs are chronic congestion, hemorrhages, infarctions, lobar pneumonia, bronchopneumonia, pleural effusions. The liver is often dark brownish in color, and presents evidences of congestion. The spleen is often enlarged, and very often contains wedge-shaped or irregularly formed infarcts, which are apt to have an ochre-yellow color, and of which the surface is beveled.

The bone marrow of the femur was examined in 3 cases. It was dark red in color, and showed active regeneration.

Of the greatest importance are the endocarditic and the glomerular lesions. In the course of the studies we found that when the mitral valve is involved[1] in cases of subacute bacterial endocarditis (and it is involved in the larger

[1] We have found this disease to occur very rarely in the right side of the heart.

number of cases), there is a tendency for the vegetations to spread up on the left posterior wall of the auricle more than on the valve itself. Often the chordae tendineae which are attached to the posterior flap are covered to a greater or less extent by vegetations. Nearly always the anterior flap is also involved, and here the vegetations tend to grow down over the chordae. Not uncommonly the chordae are ruptured, the torn ends at times being massed together by vegetations at the edge of the flap; or the lower ends may be

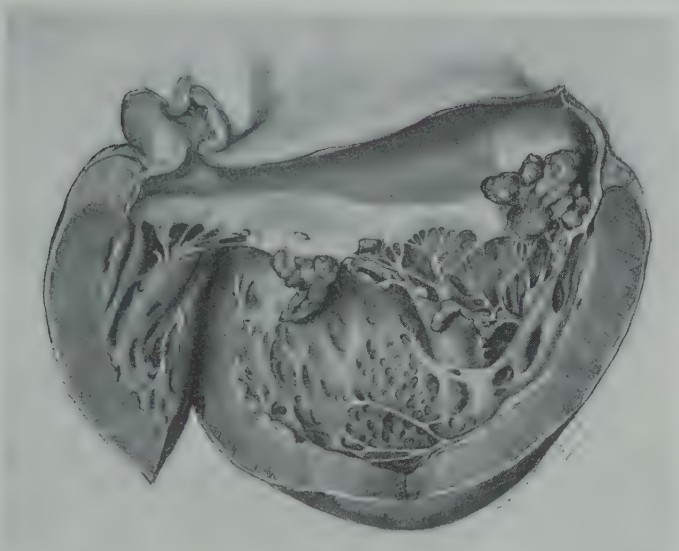


FIG. 91. Subacute streptococcus endocarditis in bacterial stage. Involvement of anterior flap of mitral valve, posterior flap and wall of auricle, and chordae tendineae. Lesions typical. Scattered areas of calcification.

found lying loose near the papillary muscles. The vegetations are yellowish, greenish, pinkish, or reddish in color, and vary much in size in different cases. As they grow older they become firmer, and assume a more grayish color.

When the aortic valves are involved, the lesions are not usually characteristic, and their extent is most variable. There may be only a small vegetation on one or more of the flaps of the valve. On the other hand, there may be enormous green masses that must block the orifice to a greater or lesser

extent. The vegetations have a tendency to extend down over the endocardium at the position of the septum membranaceum, and over the ventricular aspect of the aortic flap of the mitral valve and down over the chordae tendineae. At times they have a stalactite-like appearance. There may develop an aneurysm of the aortic flap of the mitral valve. Ulceration of the aortic valve at times occurs; ulceration of the mitral flaps is rare.

A study of the frequency of involvement of the valves, the left auricle, and the chordae in the heart from 34 cases of the disease was made with the following results:

	Cases
Auricle, mitral valve and chordae	17
Auricle, mitral valve, chordae, and aortic valve	5
Auricle and mitral valve	2
Mitral valve and chordae	1
Aortic valve only	3
Aortic valve, chordae, and aortic flap of mitral	4
Mitral valve, chordae, and aortic valve	1
Auricle, mitral, and aortic valves	1
Total	34

The auricle was involved 25 times, the mitral valve 27 times, the chordae 28 times and the aortic valve 9 times. The frequency of the involvement is very striking.[1]

The lesions which we consider so characteristic of this group of cases we did not find in a large series of cases of acute endocarditis due to the streptococcus, pneumococcus and staphylococcus. In our own cases of acute gonococcus endocarditis we did not find similar lesions. But through the kindness of Dr. Asch, of New York, I saw one case of gonococcus endocarditis, apparently of three weeks' duration, in which the lesions resembled those seen in the cases under discussion. There were not present the symptoms seen in the typical cases of subacute bacterial endocarditis, nor the glomerular lesions which we shall soon describe as being characteristic of the disease.

[1] Acute pericarditis has not been observed in our cases. In the heart muscles of these cases Aschoff bodies were not found, except in one complicated by verrucous endocarditis of the mitral valve of the type usually seen following typical acute rheumatic arthritis.

As in the group of 34 cases tabulated, only 1 was due to the influenza bacillus, and the other 33 cases to the endocarditis coccus, the lesions must be considered characteristic of infection by these cocci. There are occasional notes in the literature on this subject, particularly by Osler and Harbitz.

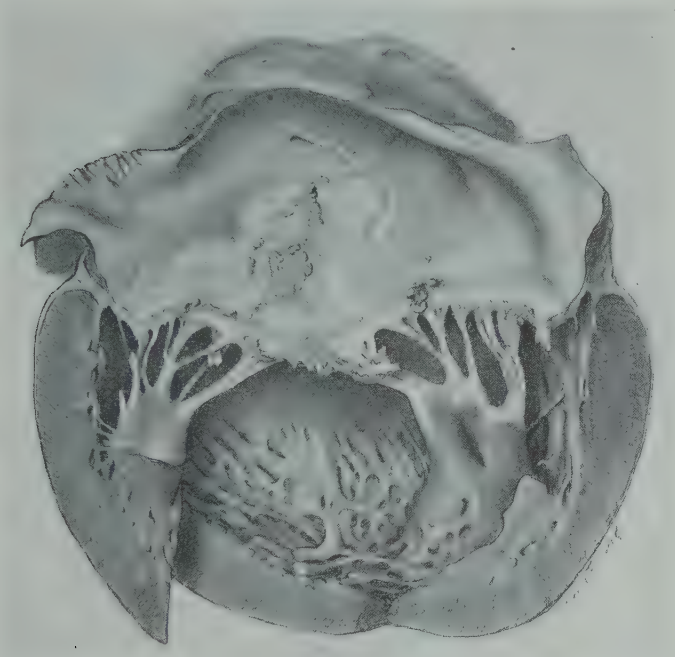


FIG. 92. Subacute bacterial endocarditis, bacteria-free stage. The lesions are almost healed, showing calcification in places.

After I had made these observations, I found, in 1909, in the heart of a man in whose case a clinical diagnosis of uremia had been made, lesions which appeared to me to represent the healed form of the lesions I had previously found in cases of infection by the endocarditis coccus. There were extensive lesions on the wall of the auricle in a state of organization, and the chordae tendineae attached to the anterior flap were found torn, thickened, fibrous and in part calcareous. Shortly after this, two other cases came under observation in

the hospital, in which the diagnosis was made of chronic valvular disease and uremia, and in these cases also similar lesions were found at the post-mortem examination. My opinion that these represented healed lesions of cases of subacute bacterial endocarditis (in all probability due to the endocarditis coccus) was confirmed by Dr. Baehr's studies of

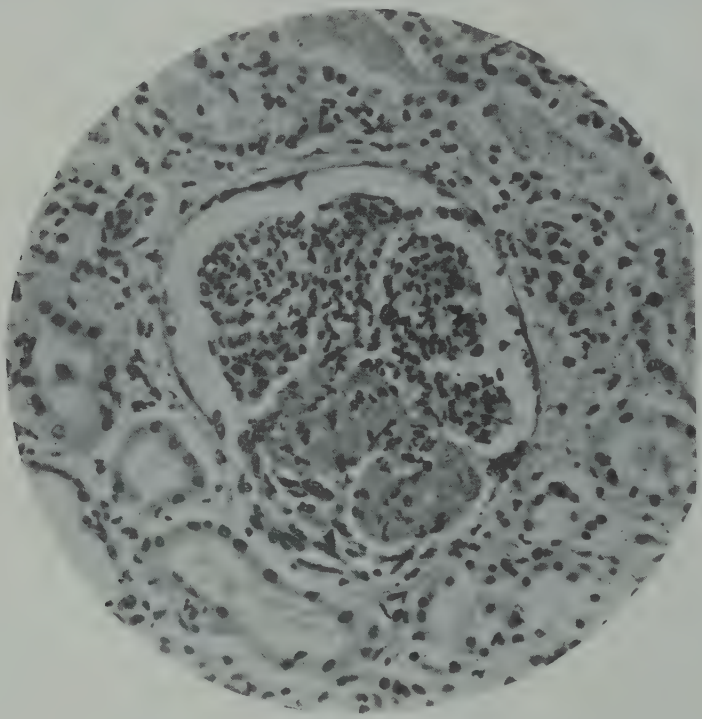


FIG. 93. Glomerular lesions of subacute bacterial endocarditis. Early stage showing destruction of a large segment. In the capsular space at the site of the lesion is seen the swollen epithelium which has proliferated and desquamated from Bowman's capsule. Stained with hematoxylin. (After Baehr. *J. Exper. M.*, 1912, xv.)

the kidneys which I had collected from cases of subacute bacterial endocarditis. Baehr investigated the kidneys of 25 cases due to the endocarditis coccus, which were still in the bacterial stage, and found the lesions described by Loehlein in 23. In 2 other cases, one an infection by the influenza bacillus, and the other the case of gonococcus infection

already mentioned, the lesions were absent. In the kidneys of 54 cases of endocarditis due to the ordinary streptococcus, staphylococcus, pneumococcus and the gonococcus, no such lesions were found. The lesions may, therefore, be considered characteristic of infections of the heart valve by the endocar-

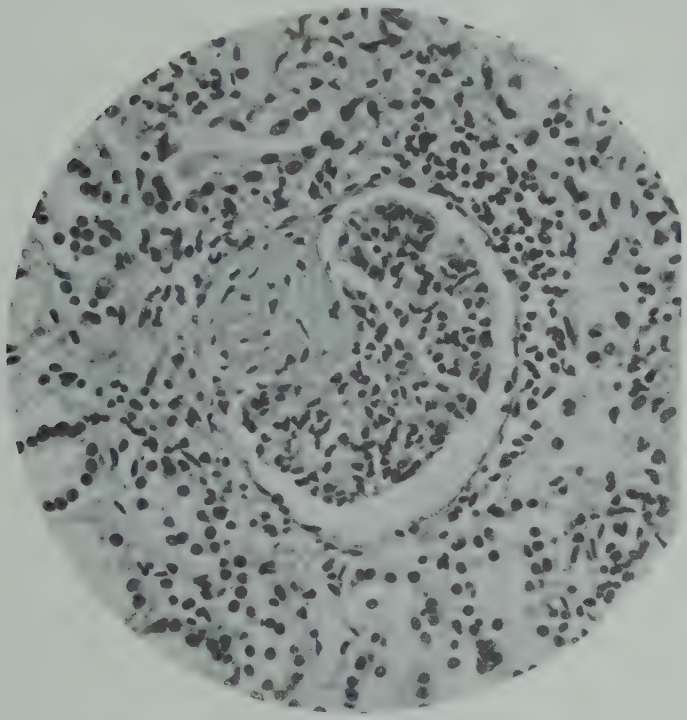


FIG. 94. Glomerular lesions of subacute bacterial endocarditis. Old lesion. The typical picture after organization has occurred. The hyaline wedge is covered on its inner aspect by epithelium which is continuous with the peripheral layer of Bowman's capsule. In the adjacent interstitium is a small collection of round cells. Stained with hematoxylin. (After Baehr, *J. Exper. M.*, 1912, xv.)

ditis coccus. Studies made by Thalhimer and the writer since the time of Baehr's publication have given similar results.

A description of the glomerular lesions will be found in the paper by Baehr (similar observations have been published by Gaskell). In a brief way it may be stated that these glomerular lesions are embolic in nature, the emboli

consisting of plugs of bacteria. As a rule, only part of a glomerulus is involved, but occasionally the whole glomerulus. The involved part undergoes necrosis, and eventually is organized.

The percentage of glomeruli involved varied from 2 to 75 per cent in our cases. If the lesions are very extensive, a secondary contracted kidney may result. Apart from this, it can happen that the embolic lesions are not present, but the patient has an atypical form of glomerular nephritis. This was found in 2 of our cases.

Acute diffuse glomerular nephritis is infrequent in the active as compared with the bacteria-free stage of the disease. Our records indicate that the latter class of cases shows a subacute or chronic diffuse glomerular nephritis at least fifteen times as often as a diffuse lesion is found in the cases in the active stage.

Inclusive of the 3 cases above described, I have, up to the present time, had the opportunity of observing over 50 cases in which the patients suffered from a subacute bacterial infection of the valve, and in which the bacteria disappeared spontaneously. In the depth of the lesion scattered bacteria may be present, as in other healed or healing conditions of bacterial origin. At the autopsies of the fatal cases there were found bacteria-free lesions of the endocardium in various stages. In many cases the lesions were entirely organized. At times they were almost entirely calcareous. In nearly all the cases, typical embolic glomerular lesions in the healed stage were present. In the cases in which these were not found there were present during life the typical tender nodes or other clinical phenomena that made the diagnosis definite.

The spleen is often very large in cases in the bacteria-free stage, and there may be found a striking enlargement of the Malpighian bodies with a hyperplasia of the pulp. The bone marrow has been examined in a number of cases, and there was usually found an active regeneration of the marrow.

In the cases that had calcareous masses in the valves, aneurysms were sometimes produced, usually at the root of the aorta, in the mitral valve or in the peripheral arteries.

I have elsewhere given definite proof that besides the typical embolic infective type of aneurysms there is an embolic non-infective type.

We will now proceed to a review of some of the clinical features in the bacteria-free stage.

Duration. This is very difficult to estimate, as most of the cases were not seen in the bacterial stage. But I have evidence to show that it can be even as long as two and a half years. As stated above, there are a few records in the literature of cases which became bacteria-free under observation, and remained well except for the presence of a previously existing valvular lesion. But the period of observation was not very long, and no later reports have been issued stating whether the patient developed any of the symptoms which we will mention as occurring in the bacteria-free stage, and by means of which we have been able to recognize clinically the comparatively large number of cases forming the basis of this presentation.

Previous History. In one case there was a history of scarlet fever at an early age. In others there were previous attacks of rheumatism and a valvular lesion. In some cases the patients had suffered for a longer or shorter time from palpitation, dyspnea, pain in the precordial region and cough, with or without expectoration. In a few of the cases there was a history of an attack of fever, pain in the joints, palpitation and dyspnea a number of months before the patient came under observation. It is proper to suspect that these attacks, in some instances, represent the bacterial stage of the disease.

In most of these cases the mode of onset was with cough and palpitation, with or without hemorrhagic expectoration. In others there were chilly sensations, fever, sweats, joint pains and swelling of the legs. In one case, pain in the thigh was the first symptom, and in another, severe pain in the sacral region. Vomiting with headache and dizziness was the initial symptom-complex in a couple of cases. In one case, epistaxis, and in another, frequency of urination first appeared.

The temperature does not run nearly as high in the bacterial stage as in the bacteria-free stage. It may be high

in cases seen (apparently) shortly after the bacterial stage has ended, or if complications develop, or an intercurrent disease. Embolisms may cause a marked rise of temperature, and at times, chills. In many cases the temperature is practically normal during almost the entire period of observation.

As a rule, no new murmurs develop during the course of the disease.

The renal phenomena are of the greatest importance. Macroscopically, hemorrhagic urine may occur, due to infarctions. Erythrocytes in small numbers are found in the urine in some of the cases, but not with the regularity or persistence with which they are found in the bacterial stage. Albumin and casts are found in some cases from time to time, and in others there is definite evidence of a more or less marked progressive disease of the kidneys. Among the cases in the latter group there is a form of disturbance quite remarkable, because the patients in this group pass large amounts of urine (up to 2750 c.c.), with a high specific gravity (1028 to 1032). I do not know that such a complex has been previously described. Symptoms due to renal insufficiency are quite prominent in a number of cases. In some they dominate the clinical picture.

There is usually present a more or less marked anemia, usually of the secondary form. The hemoglobin was in some cases as high as 65 per cent; in others as low as 20 per cent. Nucleated erythrocytes were never found. It is curious that this anemia should be progressive, even in the bacteria-free stage. The bone-marrow changes have been described. In some cases the leucocytes were diminished; occasionally the polymorphonuclears were decreased, even to 36 per cent. Histiocytes are found in only very small numbers.

The spleen was palpable in nearly all the cases; at times it was enormously enlarged. In all the cases except one, in which the spleen was not palpable, it was enlarged at the post-mortem examination, at times very much so.

Petechiae were found at one time or another in about half the cases. As a rule, few were found as compared with the number found in cases in the bacterial stage. Tender cutaneous nodules were found much less frequently. In the first 21 cases they were present only three times.

Sternal tenderness seems to be more marked in the bacteria-free than in the bacterial stage. It was found in nearly all the cases in which it was looked for. The long bones were usually not tender.

Sweats and loss of weight were not as frequent in the bacteria-free stage as in the bacterial stage. The brown color of the face, which was described by the writer in 1912 as occurring in the bacteria-free stage, was noted as a striking symptom in 9 cases. In some it developed under observation. A few of the patients who were very anemic developed a more or less café-au-lait color. This has also been observed by the writer, as stated in a previous publication, in the bacterial stage; but the marked brown color he has seen develops only in bacteria-free cases.

The only pains that occur with any degree of intensity are the joint pains, and these were observed particularly in the cases in which purpura was a symptom.

Apart from the occurrence of aneurysms of the mitral valve, the septum membranaceum and sinuses of Valsalva, which gave no clinical symptoms, there was encountered once an embolic aneurysm of the femoral artery. The patient was admitted to the hospital with a diagnosis of osteomyelitis, because of the temperature, high leucocyte count with polynucleosis and a marked swelling of the thigh. The presence of a valvular lesion and a large spleen led me to suspect that we were dealing with a case of subacute bacterial endocarditis. The post-mortem examination confirmed the clinical diagnosis, the lesions being practically bacteria-free, and, for the main part, organized.

The cardiac symptoms can be classed in two groups. We must consider first the subjective and objective symptoms, due to the valvular defect as such. Here we include the cough, expectoration, palpitation, arrhythmias, pains in the chest, congestion of the viscera, edema of the legs and hemorrhagic infarction of the lungs.[1] All these symptoms may occur in any case of valvular disease.

In the second group we include the embolisms, petechiae, purpura,[2] embolic aneurysms and tender cutaneous nodules.

[1] This can, of course, at times be the expression of a hemorrhagic tendency.

[2] These are more often than is generally supposed due to thromboses in the veins of the lower extremities and of the pelvis.

This second group occurs when there are friable masses in the valves or in the wall of the heart. They are, therefore, very common events in cases of bacterial endocarditis in the bacterial and bacteria-free stages, and may well be termed "endocarditic symptoms." Cerebral embolism seems to be as frequent an occurrence in bacteria-free as in bacterial cases.

Joint symptoms are much less common than in the bacterial stage. There may be joint pains or joint swellings or both. The phenomena are usually not marked, and are quite transitory.

The cause of death can be fairly definitely stated in 16 of the fatal cases. In 3 it was due to cerebral embolism; in 4 to uremia; in 2 to progressive anemia and an intercurrent pneumonia; in 1 to uremia, embolism and pneumonia; in 1 to pneumonia; in 3 to cardiac insufficiency; in 2 to progressive anemia with concomitant cardiac weakness. To put the matter in another way, we may say that cerebral embolism was a determining factor in 4 cases; uremia in 5 cases; anemia in 4 cases; and cardiac insufficiency (unaccompanied by profound anemia) in 3 cases.

The important rôle played by progressive anemia, nephritic changes and the presence of vegetations on the valves and musculature of the heart is thus made very clear.

From the description which we have given of the symptomatology of the disease under consideration, it might appear that most of the cases look alike clinically. But while it is true that petechiae, sternal tenderness, a palpable spleen and more or less marked anemia occur in most of the cases, there are certain important features, one or more of which may occur in any given case, for which a careful search should be made in all cases of chronic valvular disease. These features are five in number, as follows:

1. Marked progressive anemia.
2. Brown pigmentation of the face.
3. Marked renal disease. Patients presenting this feature usually die of uremia.
4. Marked enlargement of the spleen. The spleen may be so enlarged that the case may be considered one of chronic splenic disease.

5. Phenomena due to the presence of vegetations on the valves. These are what I call endocarditis symptoms. They consist more or less of elevation of temperature occurring from time to time, occasional joint symptoms and embolisms (including petechiae, tender nodes and embolic aneurysms).

It would be of great interest to discuss the differential diagnosis of subacute bacterial endocarditis from rheumatic fever, and particularly the differentiation of cases of valvular disease in which there is a superimposed bacterial endocarditis in the bacteria-free stage from valvular defects in general, and from valvular defects complicated by various other conditions. Space, however, will not permit me to go into these and a number of other very interesting questions. Therefore, I shall conclude with a short discussion of the little that is known concerning the therapeutics of this disease.

Various types of vaccines and serums have been used in the treatment of the disease. These include stock streptococcus vaccines, non-specific vaccines, autogenous vaccines, dried extracts of vaccines, sensitized vaccines, antistreptococcus serum, serum of human beings and animals previously immunized against the organisms isolated from the patient's blood, blood of immunized human beings and convalescent serum.

A few cases of recovery have been reported in which vaccines were held accountable for the favorable outcome. Latham and Hunt reported a case in which recovery was attributed to the use of an autogenous vaccine administered by mouth. Hemsted reported a case with recovery in which a variety of vaccines and serums were employed. Horder tried vaccine therapy in 6 cases due to anhemolytic streptococci, and in 2 due to the influenza bacillus. In 2 of the streptococcus cases there was some reduction of the fever and improvement of the general condition, but neither patient recovered. Billings, who reports a case of recovery, considers the vaccines and serums of no value. Our experience in a large number of cases has, however, not been encouraging. There is no proof that serums or vaccines have helped in any way to bring about recovery in patients suffering from this disease.

A large number of drugs has been used in an effort to cure the disease. Among them are the colloidal metals, especially those of silver, e.g., collargol, argochrome, dispargen, mercury compounds, arsenical derivatives, phenol, formalin, the sulphocarbolates, quinin, and the salicylates. These drugs have been administered orally, subcutaneously and intravenously. An effort was made by Epstein to treat the disease by the intravenous injection of colloidal iodin. Although the drug was found to have no curative value, the blood could be freed of bacteria for a period of four days. It must be remembered that after a febrile reaction, with or without a chill, produced by the intravenous injection of any solution, the bacteria may disappear from the blood for one to three or four days.[1]

Of all the drugs mentioned, the arsenical preparations seem to be worthy of a further trial. Lorey ascribes the recovery of his patient to the intravenous injection of salvarsan and arsacetin. One of our cases of recovery was treated with salvarsan. So many other cases have, however, been treated by repeated injections of salvarsan without result, that there is no proof as yet that the drug is of any value in this disease.

The results of Capps with sodium cacodylate are worth following up. We ourselves have as yet found no definite results. Capps reports the use of this drug in 8 cases with positive blood cultures, and of these 6 are still living. One patient is well after eleven years, 2 patients after five and five and a half years, respectively, 1 after two years and three months, and 2 patients were improving after five and six months of treatment, respectively. The patients who recovered seem to have suffered from the disease in a rather mild form, but, none the less, the large percentage of recoveries makes the method of therapy worthy of further trial. Capps used daily intravenous injection of sodium cacodylate in doses of from 1 to 5 grains, the average dose being 3 grains. The solution should be freshly prepared every week or ten days from a reliable preparation of the drug. The treatment should be kept up for a number of months if

[1] In the studies which we have thus far made, we have not observed definite results from the administration of mercurochrome, gentian-violet or acriflavine.

necessary. If diarrhea develops, the drug should be discontinued.

Transfusions are of value not so much for any specific bactericidal effect of the injected blood as for the general improvement which results in the patient's condition. It acts only in so far as it combats the anemia. The point at which we think the anemia becomes important is when the hemoglobin falls below 60 per cent. Space will not permit me to discuss the amount of blood that is to be used, nor the preparations sometimes necessary to be carried out before the transfusion is done. It must be remembered that many of these patients are suffering from cardiac insufficiency, and therefore, it is important not to introduce too much blood. I will not at this time take up the question of which methods of transfusion are to be employed.

Severe decompensation is not frequent in the active stage of this disease, but when symptoms of myocardial insufficiency develop, the principles underlying the therapy are those applicable to the same condition occurring in the course of other disease. The general measures useful are those employed in the treatment of any prolonged infectious disease, such as tuberculosis. Absolute rest in bed should be enjoined when the fever is high. It is important that the patient be very well nourished. It is unnecessary to discuss the treatment of the fever, as it does not vary in any way from that employed in other forms of febrile disease in connection with insufficient heart. The indications for treatment in the bacteria-free stage are those necessitated by the anemia, the cardiac insufficiency and the nephritis.

At another time I shall take up the question of the operative indications that may arise in the course of subacute bacterial endocarditis. On the present occasion I wish to emphasize the point that purulent infections practically never develop, and that, therefore, one should be most cautious in entering upon an operation with such an indication in mind. Patients who are suffering from this disease have been subjected to unnecessary operations for supposed liver abscess, subphrenic abscess, abscess of the spleen, or appendicitis. It has happened that a patient has had a kidney unnecessarily removed because an infarction produced symptoms leading the

clinician to believe that the patient was suffering from tuberculosis of the kidney or pyelonephritis.

Sir Thomas Horder was the first to emphasize the importance of prophylactic measures. Inasmuch as the great majority of cases are due to superimposed infections of valves which are the seat of previous rheumatic or syphilitic disease, the direct problem becomes one of the prevention of rheumatic fever and syphilis. The more direct consideration is the prevention of subacute bacterial endocarditis in patients who already have valvular disease. The problem of prophylaxis should be directed particularly to the cases in which the valvular lesion is trifling, or at the most, of moderate severity, for these are the patients who are most susceptible to the disease. These individuals should keep themselves under observation. Foci of infection representing possible portals of entry for the anhemolytic streptococci and influenza bacilli should be removed. The patient should be examined at regular intervals so that infections which develop later can be eradicated. It will be worth while making a thorough study of the value of prophylactic vaccination.

BACKACHE*

W. Russell MacAusland, M.D.

BACKACHE, because of its prevalence and its many etiological factors, is a subject of great interest to the entire medical profession, and any discussion that aids in its diagnosis and treatment is warranted. Like many medical problems it has been approached too frequently from the standpoint of the specialist. The orthopedist, urologist, gynecologist, psychiatrist, each has interpreted the cause of backache from his respective point of view. The orthopedist who is called upon to treat a large number of cases of backache should appreciate the variety of factors giving rise to the pain, and should realize that a wide knowledge of clinical conditions often is required for the solution of the problem. Backache, like arthritis, if approached with a narrow vision, not only may be treated without relief, but actual damage may be done to the patient by the removal of the so-called foci of infection. Undoubtedly, many laparotomies have been performed without relief because the pelvis alone was considered. On the other hand, many types of retentive apparatus have been employed in cases where the lesion was entirely or largely surgical. Therefore, in order that the proper treatment may be advised, a very thorough investigation of all possible etiological factors should be made. The surgeon who does not use every means at his disposal, is bound to be unsuccessful in the treatment of a large proportion of his cases. On the other hand, it is my opinion that when great care is used, the treatment of backache is satisfactory.

GENERAL SURGICAL, MEDICAL AND NEUROLOGICAL LESIONS, WITH REFERRED BACK PAIN

Genito-urinary System. It has been demonstrated many times that the following conditions may frequently be associated with back pain.

*This lecture was delivered on Nov. 9, 1923.

1. Floating kidney.
2. Diseases of the kidney.
3. Perinephritic disease.
4. Uretal conditions.
5. Deformities and displacement of the pelvic organs.

In most cases it is possible either to prove or to rule out any of the above conditions. If present, they may be accepted as a definite factor, but it should be borne in mind that even when found, they may be of only little importance in the causation of back pain.

Prostatitis and spermatocystitis have been considered definite causes by Baker,[2] Young, Geraghty and Stevens,[84] and Smith.[45] Therefore, an examination of a man is not complete without determining the condition of the prostate gland and its adnexa.

In women, malposition of the uterus has long been regarded as a potent cause of backache. Gradually, however, the old theory of pressure of the retroposed uterus on the nerves has been discarded and the more advanced gynecologists are admitting that pelvic disorders are not the direct causes of backache, but that they are usually concomitants. Graves,[17] Cooley,[9] Brooke,[6] Reynolds and Lovett,[38] Sever[44] and many others emphasize the association between pelvic conditions and back pain. Phaneuf[35] seems to me to have expressed the most reasonable view on the subject, namely, that the ordinary deformities of the uterus, such as retroversion, anteflexion, etc., are not causes, but that the heavy, boggy, mildly infected uterus sinking low in the pelvis, may definitely cause back pain by pulling on the nerve structures. A heavy, subinvoluted uterus represents the type of lesion that may give rise to back pain.

Cancer in the pelvis might also bring about a similar condition, although in such cases there is the possibility of the involvement of the nerve sheaths, and even of the nerves themselves.

Backache associated with menstruation seems to me to represent a combination of the factors: nerves, fatigue, and the laxity or susceptibility of the pelvic joint to strain.

Abdominal Conditions. Abdominal conditions associated with back pain are very numerous, particularly those repre-

senting infections, such as the infected gall-bladder, appendix, etc. The dragging of a large scrotal or abdominal hernia may also be accompanied by back pain. New growths should, of course, be considered separately. Metastases of the spine take place with such frequency that actual local disease in the spine must be considered in every case, and such a condition will be treated under lesions of the spine.

Neurological Lesions. The neurologist finds many conditions giving rise to back pain. Cases that present definite diagnostic signs of irritation or degeneration in the central or peripheral nervous system should come directly under his care. Those which represent neurasthenic, functional or hysterical conditions, should be examined with great care, as frequently such cases are malingering. In my opinion such a diagnosis is rarely justified.

Focal Infections. In regard to focal infections as a cause of backache, I believe they act always through intermediate conditions, the most common of which is arthritic infection. Focal infections, therefore, may be considered under the treatment of arthritis, the well-known cause of back pain. I do not wish to belittle the importance of focal infections, but I do wish to emphasize the need of giving attention to the intermediate condition that the infection produces, as well as to the eradication of the focus itself.

BONE AND JOINT CONDITIONS WITH REFERRED BACK PAIN

Static and Postural Lesions. The term "static" implies an alteration in the relation that the different parts of the body bear to each other. The human body works always in fixed lines and planes, with certain variations and limits of motion. Checks to the motion of the back are ligaments about the lower end of the spine which are particularly strong and relax only under extreme stress and strain. If abnormal positions are maintained for a sufficient, length of time, these ligaments may be constantly strained, or the spine or other structures may be affected, and static backache result. Constant postural and static strain may affect the back to such an extent as to cause actual arthritic changes in addition to backache (Fig. 95). If a chronic condition persists, the postural defect may progress to actual

displacement of the last lumbar vertebra. This may be demonstrated by lateral *x*-rays of the spine (Fig. 96). The displacement causes a pinch in the posterior vertebral body and is responsible for much of the pain, arthritic changes and referred leg pain occurring in this type of case.

Postural changes causing backache may be classified as flat back, lordosis, round shoulders, faulty position in the

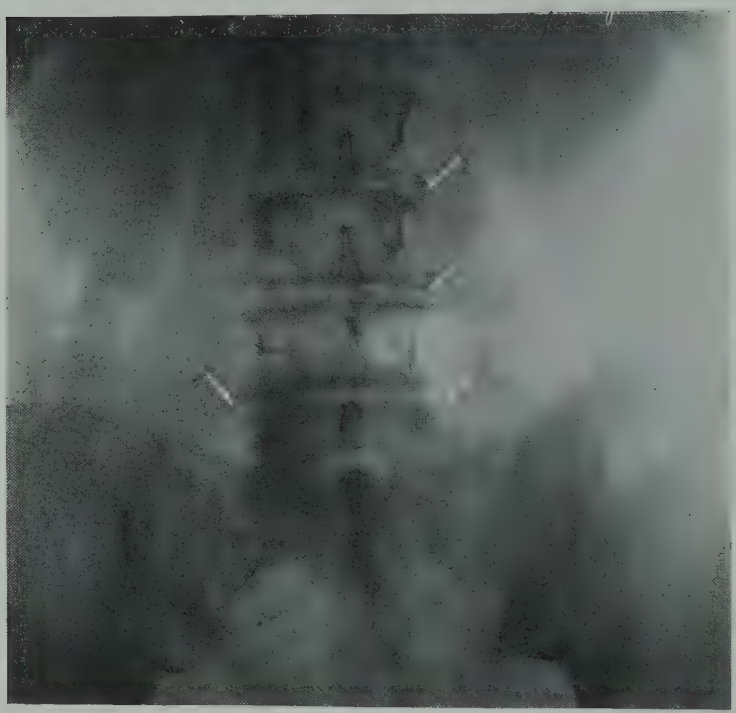


FIG. 95. Showing arthritic changes of the spine.

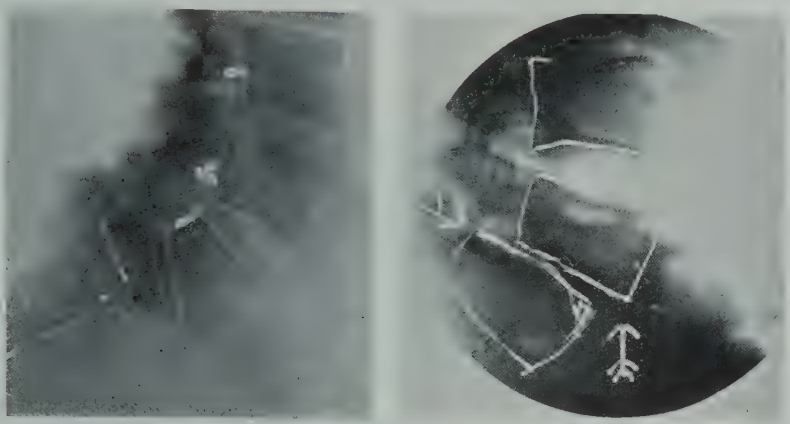
back itself, or they may be secondary from abdominal ptosis or overweight.

The static defects are chiefly due to flat feet, knock-knees, short leg, congenital hip and scoliosis.

Many of the postural defects develop from assuming a definite position in occupation or from performing constant rhythmic movements involving the spine.

A great deal has been written upon the recognition of this static element. In 1910, Reynolds and Lovett[38] propounded

the "static" theory that backache is due to the forward displacement of the center of gravity of the body and that the undue strain on the posterior muscles and ligaments causes the pain. Goldthwait and Osgood,[16] Brooke,[6] Dameshek,[11] Kuth,[24] Woodbury,[47] Ogilvy,[30] Graves,[17] Marshall,[28] Kosmak,[22] O'Ferrall,[29] have written upon the importance of this static disturbance.



A *B*

FIGS. 96 *A* AND *B*. Showing forward displacement of the fifth lumbar vertebra.

LOCAL LESIONS IN BONE, MUSCLES AND LIGAMENTS

Infections. In my experience, arthritis of the spine has proved to be the most frequent cause of back-pain conditions. Definite pathological symptoms are found, which, for the most part, are amenable to proper treatment. Arthritis of the spine is practically universal and may be demonstrated in the spine of almost every person over forty years of age. Arthritis of the spine, as of other joints, may be of various types—hypertrophic, osteoarthritic, etc. (Figs. 95 and 97). Infection from a distant foci passing through the blood may localize in a back strained from postural, static or traumatic conditions.

Limitation of motion, so characteristic of joint disease, is associated with this arthritic process. Muscle spasm, protective in nature, acting with the usual checks of motion, gives rise to an abnormal limitation.

Cancer, tuberculosis, syphilis and other diseases of the bone may also cause definite pain.

Traumatic. Injuries may result in strain or fractures. The structures involved in the traumatic processes are the sacro-iliac joints, the lumbar spine and the lumbosacral junction.

Strain. A change from the normal position produces a strain on the ligaments and muscles. A simple muscular or ligamentous strain is a common industrial complaint. The strain may be acute or chronic. Faulty posture may cause an

FIG. 97. Showing hypertrophic scoliosis.

acute strain. A dorsal position on a table maintained for a long period during operation, may produce an acute strain of the lumbar ligaments. Chronic strain occurs in cases of congenital dislocation of the hip, short leg and static defects in the lower back. The chronic types may develop into low-grade arthritic processes. Therefore, in making an examination, these static, traumatic and infectious processes should be kept in mind.

There has been much controversy in regard to the dislocation, relaxation and subluxation of the sacroiliac joints. As early as 1870, relaxation and subluxation were

associated with pregnancy. Since 1905, when Goldthwait and Osgood[16] explained their theory in regard to the dislocation of the sacroiliac joints, there has been considerable discussion. Some have agreed that slight dislocations are possible, others have demanded pathologic and radiographic proofs.

More recently it is being recognized that these displacements have been overestimated. Actually the joints between the sacrum and the ilium are the most perfectly protected in the whole human body. Four major ligaments and several minor ones give protection. In my entire practice I have never been able to demonstrate, by clinical examination or by *x*-rays, the presence of a dislocation of the joint, except in cases of severe traumatism, such as occur in automobile or railroad accidents. Relaxation and subluxation of the sacroiliac joints are infrequent, occurring only during the course of pregnancy, and they tighten up very early after delivery. It is of significance that during pregnancy, when these joints are relaxed, symptoms of back pain are only rarely present. In cases of overweight, postural conditions cause strain in these relaxed joints, and increase the symptoms and disability.

Many of the lesions supposedly of sacroiliac origin, are actually lumbosacral. This feature is often brought out in a routine examination of the back in cases of postural defects, in which there is an actual forward displacement of the fifth lumbar vertebra on the sacrum (Fig. 96).

The acute attacks that are frequently relieved by osteopathic treatment are simply muscle cramps following ligamentous strain. Such a condition is seen in cramps in the calves of the legs following overexercise and overuse.

Frequently, heat, massage and manipulation will increase the circulation and give relief.

Fractures. Infrequently, fractures occur in the region of the sacroiliac joints. In the lumbar region a crush fracture occurs most commonly at the junction of the vertebrae. Crush fractures of the articular processes and fractures of transverse process are uncommon.

Fracture, or lateral displacements of the coccyx, usually cause localized pain. In chronic cases it is largely postural in

origin, with neurasthenic conditions superimposed. In traumatic cases in which a definite fracture or displacement of the tip of the coccyx is demonstrated by digital examination and the *x*-ray, the condition is often relieved by the removal of the fractured or displaced tip. In some cases, however, pain persists after the removal. Surgery in this type of case is not to be advised.

Congenital Abnormalities. Congenital abnormalities in general should not be considered an actual cause of back pain,

Fig. 98. Showing spina bifida.

but merely a predisposing factor. These abnormalities in themselves rarely give rise to symptoms, without some extraneous cause, which is most frequently static, postural, traumatic or infectious.

Not infrequently, spina bifida or failure of the closure of the posterior arches, from which the patient has no symptoms, is found in a routine examination of the spine (Fig. 98).

Varying degrees of sacrilization, from a mere broadening or lengthening of the transverse process of the fifth lumbar vertebra to an actual antler wing on one or both sides, may be found. This sacrilization is undoubtedly of congenital

origin (Figs. 99 and 100). Ludloff[27] and Scheede[43] made the first careful study of the appearance of the structure in *x*-rays. It varies in size and in position with reference to the level of the iliacs. It may be increased in length and width and flattened out into broad, bilobed structures, tipping upward and backward. It may develop to conform with the shape of the adjacent iliacs. O'Reilly[31] studied the *x*-rays of 300 patients suffering from backache. He found three general types of

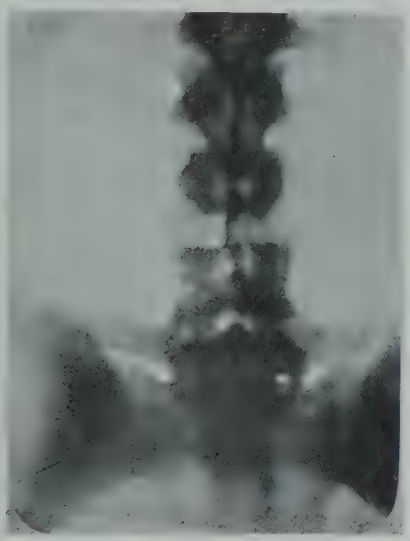


FIG. 99. Showing long transverse process of the fifth lumbar vertebra.

transverse processes of the fifth lumbar vertebra, the straight type, the fan-shaped type and the bulbous, the last being most common. It was his opinion that occupation played an important part in the development of these variations. Abnormalities of the last lumbar vertebra will cause no symptoms unless aggravated by static, postural, traumatic or infectious lesions. In such cases it is more difficult to relieve the condition. In 3 cases I have found it necessary to resort to surgical interference.

If the transverse process is so long that it comes in close contact with the iliac crest without actually being connected by a bony bridge, a bursa may develop. In some cases

pressure may result in the fusion of the tip of the process with the ilium.

Only a few cases of excision have been reported in literature. Some surgeons have tended to discourage the operation, as they claim damage may be done to the nerve structures coming out above or below the transverse process. Others believe with Epstein[13] that the postoperative treatment, not the excision itself, relieves the condition.

Blanchard and Parker[5] reported the first successful case of excision in 1915. Rugh,[42] Adams,[1] Lavieri[25] and Knox[23]

Fig. 100. Showing sacrilization of the transverse process of the fifth lumbar vertebra.

have each reported a successful case since that time. Knox reported operating on 3 other cases on which it was too early to report, but which presented good prospects.

Pathology

The pathology of these bone and joint conditions occurring in the spine does not differ from the pathology of bone and joint lesions elsewhere. The intervertebral discs may be regarded as joints. They become thinned and absorbed.

The edges of the adjoining bone become hardened and form spurs.

Swelling about a badly strained, infected or diseased articular process causes impingement on the nerve which emerges from the spinal canal through a small foramen. This foramen is practically bony in outline and confines the nerve in its bony canal. Pressure, therefore, has an effect out of all proportion to similar pressure elsewhere on nerves.

The *x*-ray pathology is easy to interpret in advanced cases, but in cases in the early stages, there is an extraordinary lack of appreciation of the slight changes.

The swelling of tissues along the spine tends to give a "washed-out" appearance to the bone and joint shadows.

There is also slight thinning of the intervertebral discs, and the edges of the vertebrae become slightly penciled, due to atrophy.

There may be a slight asymmetry of one vertebra upon another, showing that one joint has given away somewhat more than its fellow on the opposite side.

All the above signs seem to be frequently overlooked, and yet each is of importance.

Diagnosis

The diagnosis of the so-called orthopedic causes of back pain is based upon the same signs as appear in the examination of any joint.

1. Deformity.
2. Limitation of motion (asymmetrical or symmetrical).
3. Muscle spasm.
4. Definite pain or soreness on extremes of motion (when motion is forced with sudden jerk).
5. Local tenderness.
6. Local heat.
7. Local swelling.

The three latter factors may be observed only when great care is used in the examination of the lower lumbar and sacroiliac joints. Anteroposterior and lateral radiograms should be taken in erect and recumbent positions. In general, a probable orthopedic cause for back pain may be certain in any patient who presents limitation of motion of the spine,

and may be considered very probable in all cases in which this limitation of motion is accompanied by muscle spasm and local tenderness and pain.

Treatment

Bed. The bed is one of the first factors to consider in the treatment of backache. Most beds sag, whether a person is stout or thin. This sag alone may cause back pain and if continued, produce chronic back strain. This condition should be corrected so that the back is held in proper position during rest. The most convenient method is to place a fracture board between the spring and the mattress, placing a blanket crosswise on the board so that it will come under the upper lumbar spine.

All static defects, such as flat-foot, knock-knee, round shoulders and abdominal ptosis should be eliminated by mechanotherapy, correction of faulty habits, and exercises. If deformity is corrected and the proper habit of standing, sitting and lying is formed, no further treatment will be necessary.

In the infectious type of case, all existing foci of infection should be eliminated. The patient must improve his general condition by fresh air, sun exposures and good habits. Supportive treatment may be necessary, depending entirely upon the severity and chronicity of the process. In the acute stages, bed treatment is most important. As soon as the symptoms have subsided and the deformity has been corrected, some type of retentive or supportive apparatus is absolutely necessary in order to hold the correction and relieve the patient of further strain. This support may be removed when the surgeon judges that recurrence is impossible. The apparatus should be removed as soon as possible, and baking, massage and all other means employed to stimulte circulation and increase the musculature and the ligamentous tone.

If these infectious elements and all static and postural defects are eliminated, most of the back trouble, if of orthopedic origin, will be relieved.

In cases of back pain due to pelvic lesions alone, or associated with true back lesions, the pelvic, intra-abdominal or general medical problems must be treated hand in hand.

Conclusions

The most constant cause of backache from an orthopedic standpoint is low-grade arthritis. An infectious element, usually present, associated with a chronic strain, produces many of the cases of arthritis that appear in middle life.

I am certain that static and postural elements enter into almost all low back pain of orthopedic origin.

Congenital abnormalities, in themselves, are not a cause of back pain, but they may be a potential danger, making differential diagnosis and treatment more difficult.

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THE CHRONIC DISEASES OF THE JOINTS*

THOMAS McCRAE, M.D.

THE subject of chronic arthritis is one with which we have all had our difficulties, in knowing exactly how to place our own attitude in regard to it, in knowing how to classify the cases, and, most important of all, in knowing how to handle the patient.

May I devote a few moments to the question of names? You may think it is very academic to mention the terms that we employ; and yet I think we have suffered much in our discussion of chronic arthritis from the improper use of words. The particular word in this connection is "rheumatism." If you are going to have any clear idea about arthritis in your own minds a most important step at the outset is to drop the use of the word "rheumatism," and drop it entirely.

It is hardly necessary to give examples of the careless way in which the word is used. A man speaks of "chronic rheumatism." What does he mean? He usually means pain. One man talks about "rheumatism" and means rheumatic fever. Another talks about "chronic rheumatism" and means some form of chronic arthritis. The two ideas, arthritis and "rheumatism" are correlated. Then another comes along and talks about muscular "rheumatism." It cannot be muscular arthritis, can it? Another talks about sciatic "rheumatism." We do not know what he means. Therefore, I think we should drop the use of the word "rheumatism" absolutely. Rheumatic fever is a perfectly good term, but this disease has nothing to do with chronic arthritis. It never becomes chronic and never leaves any changes in the joints. In any of the joint affections why not use the word arthritis? It is a perfectly clear word. We all know what it means. Why not use it, at any rate, in our own thoughts, when we are considering the subject?

* This lecture was delivered on Nov. 3, 1922.

Perhaps I should take back what I said in one particular in regard to the use of the word "rheumatism." I believe there is one time when its use is perhaps justified. It comes in very handy, at any rate, when "mother's darling" has a gonorrheal arthritis and you are obliged to make a diagnosis and do not want to say what it is. Then it is perfectly right to use the word "rheumatism."

Recently another unfortunate usage is appearing in the literature. In a recent classification of arthritis, No. 3 was infectious arthritis: No. 4 (these were different classes) was gonorrheal arthritis. Now, if you say to that man," Is gonorrheal arthritis not infectious?" he will probably say, "Yes, but that is not the way I use the word in these cases." You cannot use a word with one meaning one day and with another meaning the next day and have any proper clearness at all. If there is any arthritis which is infectious it is gonorrheal arthritis.

Let us discuss a few points regarding chronic arthritis. We can leave out traumatic arthritis, for that does not concern us here. It is a class by itself and as a rule, gives no difficulty. Excluding that, one important thing to remember is that arthritis is never primary but is secondary to some condition elsewhere. That is, the process in the joint depends upon a process somewhere else. In other words, whatever causes the trouble in the joint is brought there, and as regards the cause, we can make two great classes, one metabolic and the other infectious. There are two or three curious forms of arthritis that one should mention: as, for instance, that which follows the injection of serum. It is a peculiar form and probably represents an anaphylactic phenomenon. The joint trouble in hemophilia is probably largely mechanical, due to hemorrhage into the joint, and is rare. The so-called Charcot's joint is probably traumatic in origin.

What comes under metabolic? So far as I know, only one form of disease, namely, gout. Those of us who have reached the half-century mark will remember that we went through a period when we heard a great deal about uric acid diathesis. We do not hear that now, or very little of it. A great deal of arthritis was supposed to be due to protein food, and people were advised to cut off the red meats, etc., but so

faras I know, there is only one metabolic arthritis, and that is gout.

Just a word or two about gout. There is no question that gout is more common in this country than we usually suppose. It is not so rare if we know enough to recognize it.

Always look at the patient's ears. In every patient with arthritis be sure he has not tophi, for patients with tophi are sometimes labeled other things.

That leaves us for special consideration the group which seems to be due to infection—in some way or other. The moment you come to a classification of chronic arthritis you meet difficulty. In some diseases we pride ourselves on making an etiological diagnosis, a pathological or anatomical diagnosis and a functional diagnosis. There is no difficulty in making a functional diagnosis in arthritis. A pathological diagnosis is usually comparatively easy, especially since we have the *x*-ray, but an etiological diagnosis is another story. It is very evident that if you can make this you settle the question. The moment you have the causal organism settled you have a clean-cut diagnosis. In many conditions we have a more or less definite clinical picture, as in typhoid fever or scarlet fever. But in the joints one organism may give many kinds of change. Take the gonococcus, for example, which may give many different results. On the other hand, the same results may be given by many different causes. So that while sometimes the pathological picture is fairly definite, it not always so, and therein lies one difficulty. We all know the classification of gonorrheal arthritis, as seen by the genitourinary man, as seen by the physican and as seen by the surgeon or the orthopedist. These are three different forms of the disease, and the three men regard the disease differently because they see different pictures of it. The genitourinary man sees many cases in which it is very slight. The physician sees the midway cases, and some bad ones. The surgeon usually sees only the severe ones. Out of that has grown the belief of many that gonorrheal arthritis is a monarticular affair. I have never seen a case in which gonorrheal arthritis was monarticular. It occurs at first in two or more joints but usually "settles" in one joint, and by

the time it gets to the surgeon it is also almost always confined to one joint.

Then we have another group in which we have an acute infection and there is no question about the etiology. For instance, a patient with pneumonia has an arthritis due to the pneumococcus, which is a perfectly definite group.

There is another group due to infection. For example, a patient has an attack of acute tonsillitis and following that an arthritis. We generally speak of that as metastatic. This is usually acute but it may advance to a chronic condition.

We have remaining a very large group of chronic arthritis, in which the etiology is often in doubt, but the clinical picture is more or less definite, and you may put those down for the moment as chronic arthritis, a safe term. My own costom is to employ the term arthritis deformans.

Now, you may say that this is not a very good classification. Of course, you are perfectly right, but it seems the best we can do. In some we have a definite etiology and in some we have not. We have to leave them as a more or less indefinite group of chronic arthritis, but clinically, they give a fairly definite picture. Sometimes one may have difficulty in deciding whether a case of chronic arthritis is gonorrheal or whether it belongs under arthritis deformans. As time goes on we may be able to classify these under definite etiological headings, but at present we must regard them as chronic arthritis.

Comments During Examination of Patients

There are some patients who illustrate some of these points and my effort will be to pick out the essential ones.

1. The first patient, a female, is aged forty. She has arthritis in the knee-joints, the ankle-joints, one hip-joint, the elbows and wrist-joints. Her past history is apparently negative. As for the present illness, three years ago she began to have pain in both shoulders and in one foot. Then subsequently the wrists, elbows and both knees were painful. She has had "flare-ups," that is, the process quieted down and then, a little later, became more or less acute.

I desire to emphasize the method of onset in many cases that ultimately become chronic. In a considerable number,

perhaps 40 per cent, the onset is acute with involvement of many joints. There are two main forms: one in which there is simultaneous involvement of many joints, and the other (which we have here) in which the disease begins in one joint and then involves another, never extremely acute, but always tending to advance.

You see that the joints do not show a great deal of deformity. One should emphasize the fact of the finger-joints being involved. A little point worth keeping in mind is that involvement of the thumb-joint nearly always means arthritis deformans. The fingers are very characteristic. One finger shows marked so-called pod-shaped thickening at the joint, and slight deformity at the terminal joints with a little flexion.

Our problem is to help this patient; to do our best to restore her as nearly as possible to a normal condition. But the most important lesson in this group is prevention. If you call this "rheumatism"—? You see a patient with this form of arthritis, call it "rheumatism" and put her on salicylates, or whatever your favorite treatment may be, and rest content. It is hard on the patient. There is the knee-joint. This patient has fair movement there and it does not hurt her very much. At first sight you may ask why the bony parts are so much enlarged; but it is the periarticular structures which are enlarged, and not the bones. The capsule of the joint is considerably thickened. There is some tenderness, particularly on the inner side of the joint. There is a certain amount of effusion in the left knee.

How would you classify this patient's disease? The joint condition is arthritis deformans, but there are other points to note. The patient is more or less emaciated and looks sallow. The hemoglobin is down to a little over 60 per cent; the red cells are about 4,000,000.

What is responsible for this arthritis? I do not think there is any question but that it is due to infection of some kind. You may ask, can the gonococcus do that? It might, but this does not look like it. That rarely damages a number of joints; and gonorrheal arthritis, as a rule, does not damage fingers like that. We are dealing with arthritis deformans

which I should classify as the periarticular type. Some men call it rheumatoid arthritis.

What is back of this? Most of us consider it as due to infection somewhere. I need not dwell on focal infection. You are all familiar with that, but may I emphasize one or two phases of it? The type "above the collar" we know well. This patient has had much infection. She had pyorrhea—several teeth with abscesses at the tips of the roots—and chronic tonsillitis. Other areas, such as the sinuses, have been looked into and they are negative. Take a person over forty years of age. You will find very often that if there are infected teeth there are also infected tonsils. These two conditions often go together. It is important not to rest content with one focus, but always to look for others.

Supposing one removes this infection and the patient does not improve as one hoped. Do not forget that the joint has been injured, and removing the focus of primary infection is not going to restore a damaged joint to its normal condition. It is often said that a patient is not any better after that has been done, but the reason may be that permanent damage has occurred.

In this patient, if all the infection is removed, the chances are that she will improve greatly. She will not have absolutely normal joints, but the permanent damage will be comparatively slight.

There is another point. With infection of the teeth or tonsils, there may be infection elsewhere; and I wish particularly to emphasize the importance of infection in the biliary tract. In a number of these cases a secondary infection of that tract occurs, just as secondary infection of the urinary tract may occur. By using the method devised by Lyon, one can often get positive results in the bile.

2. The second patient, a female, is aged thirty. In childhood she had attacks of sore throat and always had a discharge from the left nostril; she had influenza at nineteen and pneumonia, followed by empyema, at twenty-one. The present illness dates back seven years, when one ankle became swollen, tender and very painful. A little later the other ankle was involved in the same way. There was apparently never much swelling, but the pain prevented her

from getting about. She improved somewhat, but later the knees, one hip joint, the shoulders and wrists were involved. These gradually became stiff.

The condition here is not unlike that in the other patient, except it is not as marked. There is a good deal of crepitus in the knee-joints with thickening of the capsule. The condition of the reflexes is important, particularly when they are much exaggerated. Very often with improvement you find the exaggerated reflexes diminishing. There is a certain amount of atrophy of the muscles. There is considerable deformity of the hands—very much the same type as in the other patient. There is hyperextension of the some of the finger points, but no great damage to the wrists.

The restriction of movement about the shoulder-joint is often muscular but this patient has a fair degree of fixation in the joint.

The cause here is infection. Nothing else produces these results. Both tonsils are infected, she has an infection in the nose, and there is also sinus infection.

As to these patients, here is one with three years' duration and there is one with seven years' duration. This case has been of the type beginning more acutely. The other has had a more or less slow, gradual progress.

How much can be done for these two patients? A great deal will depend on getting at the cause. In one, the primary cause has been removed. The question of infection elsewhere still remains. She is improving, but it is a long, slow fight. In the other patient the sinus has been drained, but the tonsils have not been removed.

What we have to do in handling these patients is to prevent further advance and repair the damage done. If we can stop the disease from going any further, most of these joints will come back to fair use. She will have some permanent trouble in the hand. It is important to try to prevent this type of deformity. If you are patient, you can do a good deal. For instance, if this patient's hand could be kept on a splint at night, not too tightly, you would keep the fingers more or less straight. It would be worth while, as it might lessen future deformity.

One point which has come up recently in regard to these patients should be mentioned. That is the question of the metabolism. I suppose this patient thirty years ago would have been told nine times out of ten to cut out meats and not to eat any acids. As far as we know, protein metabolism has nothing whatever to do with this disease. But the question has been raised as to whether disturbed carbohydrate metabolism may have something to do with it. These patients are more or less undernourished and some do not seem to have the ability to handle carbohydrates normally. Some men advocate putting them on a very light diet. Personally, I think this is but rarely indicated. These patients are both badly nourished. If they can use food, I think they should have it, for they need it. Sometimes any change seems to be of benefit. If they have been heavy eaters, cut down on their diet, whereas, on the other hand, if they have been taking a very light diet, try and increase the quantity.

With respect to treatment these 2 patients should be handled much like a patient with tuberculosis—living out of doors as far as possible and taking a full diet. They should be encouraged to use the joints in every way possible. Within limits short of severe fatigue, the more they use them the better. The anemia should be treated; for this arsenic is often useful, particularly hypodermically.

Naturally, the question arises as to the use of vaccines. They have a place, but whether as vaccines or merely as foreign protein is a question. With regard to the advertised mixtures, combinations of millions of this and that organism —I do not think you need them. You will get just as good results by using typhoid bacilli in these patients. If, however, you can get an autogenous vaccine, it is well worth while giving it a trial. We have to work out the dosage for each patient.

In the use of vaccines in arthritis, we may pursue one of two courses. We may begin with very small doses, the most minute that can be given. We talk about the number of bacilli; but that, after all, is only approximate. The dose should be increased very gradually, without attempt to get a reaction. The other method is to begin with a large dose deliberately and aim to produce a marked reaction. I do not

know of any rule to follow in a given patient. Whether in this patient minute doses with no reaction, or a large dose with a reaction, will be best, has to be determined for each one. Some patients benefit most by one method and some by the other.

3. The third patient, a male, aged thirty-seven, is a very good patient for a clinic, because there is a good deal of doubt as to what form of arthritis he has. He complains of pain in the left knee. His trouble began nine months ago with pain in the left knee, at first only on motion and not enough to prevent him from working. A month afterward, the joint suddenly became swollen, red, tender and painful, and has remained so almost up to the present. With such a history one should go very carefully into the question as to whether this was actually a one-joint affair, a question that has considerable bearing on the diagnosis. He states that the other knee-joint was also involved; so it was not a monarticular arthritis. It was never as severe in the right as in the left knee.

He has a somewhat swollen knee-joint with a little effusion. There is quite marked tenderness on the inner surface. The capsule is tender on pressure and slightly thickened. There is moderate thickening behind the knee. The other knee shows some crepitus and the joint capsule is thickened.

This process has lasted ten months in all. The right knee shows very little change, but still definite change. The left knee shows quite marked change, and he is unable to use it.

There are three things suggested in this case:

1. It may be arthritis deformans, with involvement of an unusually small number of joints.

2. Gonococcus arthritis. It is never safe to leave that out.

3. Syphilis. The knee jerks are considerably increased. The eye reflexes are sluggish, the arm reflexes are present and normal.

The question of diagnosis is not an easy one by any means. In this patient's history there is a definite statement of a primary lesion seven years ago, for which he was given active treatment. There is no history of a gonococcus infection. I do not see anything else in his history or examination that is especially important. It is a case in which, clinically, seeing

the patient for the first time, I should not be willing to pass definite judgment on the appearance of the joints.

Now we turn to the serological findings. The blood Wassermann is negative. The gonococcus complement fixation test is fairly markedly positive.

Examination of the mouth shows no tonsillar trouble. There is some nasty infection around the teeth, however. The teeth have not been examined by *x*-rays as yet.

I might say it is this or that, but it would be guessing. It is perfectly possible it is any one of the three (arthritis deformans, gonococcus or syphilitic arthritis). What more can we do that would help? Naturally, the *x*-ray might give information. That, however, in this case, does not show very much change in the bones or cartilage. The changes are mostly periarticular. The *x*-ray study does not help us.

One should do prostatic massage and carefully examine the material obtained. When one is in doubt about a joint like this, three times out of four, it turns out to be a gonococcus arthritis. I would not make a positive diagnosis on a complement fixation test. The other is the question of syphilis. I do not think there is much doubt about it even if the Wassermann reaction is negative.

What about the therapeutic test? It is often helpful. He has been taking specific treatment, and under that the joint has been improving slowly. Frankly, I would not like to make a diagnosis on that today. The fact that he has lues does not exclude the possibility that he may have something else, such as gonorrheal arthritis or arthritis deformans. This patient is unusually tender in the knee, as a very light touch causes considerable pain. I do not know how much that is worth but it is sometimes a feather that suggests a gonococcus wind blowing. It is one of the things that make one suspicious. I think it is a very good thing to show in a clinic a patient in whom you cannot make a diagnosis. Perhaps some of you are willing to make a diagnosis, but there is not enough to make me absolutely certain.

4. The fourth patient is thirty-nine, and complains of trouble in the left knee, which dates back nine years, beginning with pain. At the end of a month it was so severe that he could not sleep. At no time did "rheumatism" medicine

help him. At the end of four months the knee was red, tender and swollen to twice its natural size. The diagnosis at this time was synovitis. He was then sent to a hospital, where an *x*-ray examination revealed slight enlargement of the end of the femur. A cast was applied and kept on for two months. When this was removed the knee was ankylosed at an angle of 40°, and he walked with crutches. Another roentgenogram was made, which apparently gave a definite diagnosis. Clinically, with a condition like this, one would naturally suspect tuberculosis.

The patient had a good deal of trouble in getting the knee straightened out. He did pretty well until May of this year, when the process started up again. At this point the patient said he had the same thing as before, but that there was not much pain.

There is a certain amount of redness present, but whether that is from the arthritis or from counter-irritation is a question. There is not much thickening of the capsule. The duration, the changes in the joint and the *x*-ray study all point to tuberculosis being the cause of arthritis here.

The important thing in this arthritis deformans group is to recognize the cases early and to try to cut short the progress before there is much damage. Much can be done to prevent these dreadful late deformities. True, it needs a great deal of patience. Do not handle them unless you are willing to work hard and be patient; for the search for the source of the infection may be a long one. Do not forget that with a primary focus you may have secondary foci elsewhere.

To repeat the point made at first: if you want to help those with arthritis, try and see if you cannot get on without the use of the word "rheumatism." If you give a thing a name, you dispose of it in your own mind more or less. Very few men are willing to go on and try to get at the bottom of something which is disposed of by giving it a name. If you say chronic arthritis and do not know what it is, then you are going to try to get at the bottom of it.

RENAL FUNCTION IN CLINICAL MEDICINE*

HERMAN O. MOSENTHAL, M.D.

THE VALUE OF TESTS FOR RENAL FUNCTION IN THE PRACTICAL MANAGEMENT OF BRIGHT'S DISEASE AND OTHER CONDITIONS

TWENTY years ago it was thought that if a practical test for renal function could be developed, the entire problem of nephritis would be solved. The first test for renal function "as a whole" was the phthalein test, which was experimented with a great deal and found to be adequate for its purpose. However, it has been realized during the past few years that kidney function did not diminish in its entirety, but that it progressed in a manner peculiar, in a certain measure, to the individual case and according to the type of kidney disease. It has come to be appreciated that in some instances water excretion is at fault, in others nitrogen excretion, and in still others the elimination of salt. To be informed concerning these different functions of the kidney demands special tests, and without a knowledge of these facts a physician is at sea in regard to what he can do for his patient.

In certain instances, especially in regard to the advisability of operative procedures, the phthalein test still is the test for renal function of choice, because it yields all the information necessary, in the easiest and simplest way.

MECHANISM OF RENAL FUNCTION

To have a thorough understanding of the way in which the solids in the urine are eliminated, it is necessary to remember that their excretion is dependent on two factors: First, the ability of the kidney to put out the solid material; second, the power of the kidney to put out water. If either one of these is deficient, the kidney must of necessity exhibit a

*This lecture was delivered on Nov. 17, 1922.

diminished renal function in regard to the particular substance concerned. This is not generally realized, but when we speak of an insufficient urea excretion we think of the solid substance only. However, when an increase in the blood urea is present in cases of myocardial insufficiency, acute nephritis, bichlorid of mercury poisoning and others, it is the inability of the kidney to put out water that is at fault, whereas, in reality, the kidney still is able to excrete urea, and it is only the lack of fluid to carry the solid away that is to blame for the diminished renal function. Hence, in order to dissect the component parts of renal function it is necessary to have tests which not only inform us concerning the excretion of the various solid materials we are considering, but also in regard to water elimination.

Importance of Metabolism in Relation to Renal Function

Tests for renal function may be applied in clinical medicine, not only to determine the efficiency of the kidney, but to obtain information in regard to the adequacy of the patient's diet.

If the patient is allowed to carry out his daily routine and to eat his usual food while he is being "worked up," very valuable data can be had as to the proper level of food intake in any given case.

We are very prone to treat cases of albuminuria and nephritis by the wholesale restriction of protein foods, and lose sight of the fact that, in the first place, the patient has to be nourished; and in the second, in many instances the nephritis itself does not demand such drastic limitations.

As far as possible, we should be informed concerning the power of the body to eliminate and digest the proteins, fats and carbohydrates. Our knowledge concerning all these facts is possibly somewhat deficient, but we have made great strides, and we should make use of the facts at our command. Possibly thus far we have not done so to the fullest extent.

In the first place, we can make a short survey of those procedures that are of value in telling us how the kidney is functioning.

Tests Employed

A short resumé of the tests employed and their significance is as follows:

Blood Chemistry. The most available tests to determine renal function are those for uric acid, urea and creatinin. V. C. Myers and his collaborators have shown that these substances are retained in the order named above as the power of the kidney becomes impaired. A useful table which approximates the relative values of these determinations is as follows:

Table 1

Blood Chemistry as a Measure of Renal Function

Impairment renal function.....	Normal	+	++	+++	++++
Uric acid mg. per 100 c.c.....	3	3–5	6– 8	9–10	11+
Urea N. mg. per 100 c.c.....	15	15	15–40	40–65	65+
Creatinin mg. per 100 c.c.....	3	3	3	3– 5	5+
Sugar, per cent.....	.09–.11				

In addition to the substances mentioned above, the blood sugar is worth considering. This in no way is a measure of renal efficiency, but it does act as a sign of the body's ability to digest the amount of starch taken in with the food. Overeating sometimes is the sole cause for a high blood sugar. For similar reasons the urea may rise in the blood because an excess of protein has been eaten. It is obvious that the interpretation of these figures must to a certain extent be left to the physician's judgment and can only be approximated in any table.

Test Day for Renal Function. It was formerly thought that to elicit variations in the specific gravity of frequently voided urinary specimens, it was necessary to administer a test diet containing diuretic substances, such as tea, coffee, meats and salt. This has proved to be incorrect, and it has been shown that even the blandest diet, such as water only, will result in such variations of specific gravity as will serve to test the ability of the kidney to concentrate and vary the specific gravity of the urine. If no special dietary is necessary, the patient may follow his routine habits and collect his urine at frequent intervals. An ordinary traveling bag, such as I show you here, containing compartments for eight twenty-ounce, wide-mouthed bottles, provides a convenient

means for collecting the specimens while the subject is pursuing his routine occupation. Directions which have been found useful in this connection are given in Table II. The hours may be modified to suit the habits of the individual. The only possible deviation from the customary way of living is found in the order that no food or fluid is to be taken after the evening meal. The charting of the patient's meals for the day furnishes a valuable bit of information concerning his dietary habits.

TABLE II

DIRECTIONS FOR TEST DAY FOR RENAL FUNCTION

The patient may eat and drink what he is accustomed to, but he should take *neither food nor drink after supper until the test is completed.* Note time and approximate amounts of food and fluid taken at each meal, on the back of this sheet.

8.00 A. M.	Void urine and discard the urine.
8.00 A. M.	Eat breakfast.
10.00 A. M.	Void urine and place all in the labeled bottle.
12.00 Noon	Void urine and save as before.
1.00 P. M.	Eat lunch.
2.00 P. M.	Void urine and save as before.
4.00 P. M.	Void urine and save as before.
7.00 P. M.	Void urine and save as before.
7.00 P. M.	Eat supper.
10.00 P. M.	Void urine and save as before.
	All urine voided after 10.00 P. M. until 8.00 A. M., is to be placed in remaining one or two bottles and labeled 8.00 A. M.
8.00 A. M.	Void urine and place in above bottle or bottles labeled 8.00 A. M.

The criteria by which the results of this procedure are to be interpreted are simple. They are shown in Table III. Special attention is called to the fact that a concentration of urine at a high level may be due to the insufficient drinking of fluid; this fact serves to make the diagnosis of a faulty habit and leads to very useful therapeutic directions and does not militate against the usefulness of the test, as urged in some quarters, but rather enhances it.

TABLE III

NORMAL STANDARD FOR TEST DAY FOR RENAL FUNCTION

Maximum specific gravity	1020 or higher.
Degrees of variation of specific gravity	9 (less if too little water is taken).
Volume c.c. of night urine usually	400 c.c. or less.
Volume c.c. of night urine doubtful	400 c.c. to 725 c.c.
Volume c.c. of night urine abnormal	725 c.c. or more.
Urea in any specimen	Normal if 2 per cent or higher. Not necessarily abnormal if less.
NaCl in any specimen	Normal if 1 per cent or higher. Not necessarily abnormal if less.
Total amount of urea and sodium chlorid eliminated in 24 hours.[1]	A measure of the quantity of proteins and sodium chlorid ingested. Insufficient elimination must be judged by the level of urea and sodium chlorid in the blood, and by edema.

[1] The hypobromite method is satisfactory for the determination of urea; the results obtained with it are equivalent to the total nitrogen of the urine, as most of the non-protein nitrogenous constituents of the urine yield their nitrogen in this procedure. The Volhardt method has been employed for the determination of the sodium chlorid.

SIGNIFICANCE OF SPECIFIC GRAVITY IN THE CASUAL SPECIMEN OF URINE

It is not sufficiently known that the specific gravity in a single specimen of urine has a very great significance. If a single specimen is had with a specific gravity of 1020 or over, we are able to obtain certain definite information from it. This is that the power of the kidney to concentrate the nitrogenous end-products is normal in these cases. This statement has no exceptions. Consequently, one is able to draw the conclusion that such patients possess a normal renal function with two exceptions. These occur, as shown in the following table, in myocardial insufficiency and in "albuminous"

nephritis. These pathological conditions can usually be readily obtained by physical examination and routine urine analysis. Furthermore, from the urine alone these diseases can be picked out because of the very low salt concentration exhibited in the urines. It is certainly of very great value to know that renal function to excrete nitrogen is normal if the specific gravity is 1020 or higher, except in the two conditions mentioned. In them the renal function, if diminished, is curtailed in two particulars: there is a retention of both salt and water. The other aspects of kidney function are not involved in the least.

TABLE IV

SIGNIFICANCE OF SPECIFIC GRAVITY IN THE CASUAL SPECIMEN OF URINE
High Specific Gravity ±1020

Diagnosis	Sp. gr.	Album.	NaCl, per cent	Urea, per cent
Normal	1020	0	0.99	2.03
Diminished function, retention water and salt				
Myocardial insufficiency				
Extreme	1022	+	0.02	3.77
Marked	1020	+	0.25	3.08
Slight	1022	+	0.52	3.38
Acute diffuse nephritis				
	1020	+++	0.43	1.90
	1020	+++	0.83	1.50
	1010	+++	0.49	0.80
Chronic diffuse nephritis				
NaCl and H_2O retention	1025	+++	0.08	1.99
NaCl and H_2O elimination rapid				
Restricted fluids	1021	+++	0.88	1.10
NaCl and H_2O elimination rapid	1010	+++	0.34	0.81

A specific gravity of 1010 or lower is much more difficult of interpretation. It may, of course, occur in a normal kidney. On the other hand, it may be the sign of a very much diseased kidney.

The following table explains itself.

TABLE V

SIGNIFICANCE OF SPECIFIC GRAVITY IN THE CASUAL SPECIMEN OF URINE
Low Specific Gravity ±1010

Diagnosis	Sp. gr.	Album.	NaCl, per cent	Urea, per cent
Normal	1010	0	0.43	1.11
Diminished function (inability to concentrate,				
Primary contracted kidney	1010	+	0.30	0.64
Secondary contracted kidney	1010	+++	0.40	0.60
Acute nephritis eliminating edema	1010	+++	0.50	0.71
Cardiac decompensation, recovering, eliminating edema	1010	8	0.51	0.94

Polycystic kidney, cystitis, pyelitis, anemia, water-drinking habit, prostatic hypertrophy, diabetes insipidus, spinal cord lesion and bladder paralysis, etc.

Attention, however, must be directed to the fact that such a specific gravity indicates pathological activity of the urine. We now have not only a diminished ability of the kidney to concentrate salt, but also a lessened power to concentrate urea, and under these circumstances, the kidney has a compensatory action by means of polyuria. When such compensatory polyuria ceases in these cases and the amount of urine excreted is diminished, the last factor of safety which the body possesses is broken down and the case becomes extremely serious. There is one exception to the above statement: a low specific gravity often persists for some time in those patients who have been retaining a good deal of fluid and who at the moment are eliminating their edema.

TYPES OF KIDNEY DISEASE

From the point of view of renal function, nephritis may be classified in many ways. The final word on this question has certainly not been said. From the clinical point of view, a useful classification is one that divides this disease into two great groups. The first of these (Table VII) may be designated as the "silent" kidneys. The urine shows no pathological constituents on the routine examination. The disease is caused by the progressive destruction of the glomeruli

through arteriosclerosis or connective-tissue formation, and the result is that they cease to functionate without giving rise to albuminuria, blood cells in the urine, or casts. Generally, such arteriosclerotic kidneys or primary contracted kidneys are the result of an increased blood pressure, although the same process may take place as the result of some other cause besides a hypertension. The outstanding feature of this condition is the inability of the kidney to excrete solids and the compensatory polyuria which occurs. A low fixed specific gravity is characteristic of such urines and an increase in the non-protein nitrogenous constituents of the blood is present in all the advanced cases. Sometimes there is added inflammation, and then we get the so-called combination form in which albuminuria is present.

The second great group is the one which we may designate as the albuminuric type. It is characterized by inflammation or degeneration in the kidney tissue, and evidences of these two processes are found in the urine in the shape of albumin, casts and red blood cells. This type of nephritis is characterized, as far as the renal function is concerned, by inability of the kidney to excrete water and salt. It fluctuates remarkably from day to day, and whereas in one week there may be very marked edema and oliguria, the next week may see the condition absolutely reversed. Sometimes albuminuria only is present and the oliguria and edema may not be found even to a slight degree. The power of these kidneys to excrete urea is usually good unless the quantity of water eliminated becomes so small as to hinder the carrying off of the nitrogenous substances. It is this type of renal disease that Epstein especially has been treating with a high protein diet; and it is evident that from a functional point of view, such high protein feeding is justified. Furthermore, it is usually very strongly indicated because the blood condition is maintained at a normal level thereby; without such feeding these patients are likely to develop anemia, partly because of the loss of albumin in the urine.

The terminal stage of these forms of nephritis is one in which connective tissue replaces the degenerated or inflamed areas. The result is a sclerosis of the kidney, which is usually spoken of as the secondary contracted kidney, in contrast

to the sclerosed kidney following arteriosclerosis, which has been designated the primary contracted kidney. In this terminal stage of the disease, that is, the secondary contracted kidney, the renal function assumes the characteristics found in the primary contracted kidney. This is readily understood, inasmuch as the lesions in the two forms of nephritis are identical, though the process of formation of the lesions has been entirely different.

I wish to emphasize again that there are two types of renal disease, the one characterized by inability of the kidney to excrete solids, both nitrogen and salt, which inability is compensated for by polyuria; and the other, in which there is a diminution of water and salt elimination, unaccompanied by any curtailment of nitrogenous elimination.

The following table shows you how the phthalein may be diminished in a given case without there being any impaired power on the part of the kidney to eliminate salt or any of the other solids in the urine.

TABLE VI

EXAMPLES OF THE VALUE OF THE SPECIFIC TESTS OF RENAL FUNCTION, AS COMPARED TO THOSE FOR RENAL FUNCTION AS A WHOLE

Phthalein low

Male. Age 70. B. P. $175/100$

Phthalein: 22 per cent in 2 hours, and 15 per cent in 2 hours (bladder irrigated).
Blood Sugar: .12 per cent. Urea N. 8.7 mg. Uric acid 2.0 mg. Creatinin 1.5 mg.
Urine: Album. 0. Very rare hyaline cast.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	145	1020				
10–12	40	22				
12– 2	20					
2– 4	87	23				
4– 6	72	24				
6– 8	55	25				
Total day	419	....	.88	3.7	1.5	6.3
8– 8	660	20	1.00	6.6	1.5	9.9
Total	1079	....	....	10.3	....	16.2

Diagnosis: Essential hypertension (minimal degree arteriosclerotic kidney).
Renal Function: Normal except for phthalein.
Metabolism: Water intake low. NaCl intake high.

In this man it was thought that possibly there was enlargement of the prostate and hence a diminished excretion of phthalein. Therefore, a second determination was made, at the end of which his bladder was irrigated to insure the proper elimination of the dye. The phthalein would indicate a very markedly diminished renal function, as the other tests indicate normality (Table VI). In this patient no attention was paid to the phthalein, and the subsequent course of events has entirely justified this point of view. This man, at the end of three years, is doing well and is not suffering from renal insufficiency.

EXAMPLES OF RENAL FUNCTION IN VARIOUS TYPES OF KIDNEY DISEASE

TABLE VII

NEPHRITIS: A CLINICAL CLASSIFICATION FROM THE POINT OF VIEW OF RENAL FUNCTION

Essential hypertension	*Acute albuminuric nephritis*
High B. P., normal renal function (heart, kidney and cerebral arteries become affected)	Retention salt and water
↓	↓
Arteriosclerotic kidney	*Chronic albuminuric nephritis*
(*Primary contracted kidney*) Renal function diminishes	Retention salt and water at intervals
↓	↓
Combination form	*Secondary contracted kidney*
Albuminuria added	Renal function diminishes

It should be noted in regard to urea as a measure of renal function, that any figure above 65 mgm. of urea nitrogen in the blood indicates the severest type of renal insufficiency. We have all seen cases in which the urea nitrogen rises well over 100 mgm. The fact is that when the urea nitrogen reaches a level of 65, then the factor of safety which the kidney possesses before complete cessation of function manifests itself is very small, and the rise from 65 to the neighborhood of 150, which may be considered a terminal stage, is one that usually takes place with great rapidity. Therefore, the urea nitrogen of 65 or over demands active therapeutic interference as far as the diet is concerned, and every effort should be made at this point to adjust the intake of the protein food in such a way as to keep the level of the urea nitrogen below 65.

TABLE VIII

ACUTE NEPHRITIS

Male. Age 38. B. P. 142/76 to 160/90.

Blood Sugar: .14 per cent. Urea N. 27.4 mg. Uric acid 2.9 mg. Creatinin 1.9 mg.

Urine: Album. 0.5 gm. per L. R.B.C. 3-plus. W.B.C. 3-plus. Coarse granular and epithelial cell casts.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	100	1012				
10–12	230	12				
12– 2	170	15				
2– 4	240	12				
4– 6	170	12				
6– 8	160	10				
8–10	150	12				
Total day.....	1220	....	.06	.73	1.3	15.9
10– 8	1300	11	.16	2.08	1.4	18.2
	2520	....	...	2.81	...	34.1

Diagnosis: Acute nephritis.

Renal Function: Low fixed Sp. Gr., polyuria (eliminating edema? Hyposthenuria), high blood urea (previous retention? protein destruction?).

Metabolism: Low NaCl output (NaCl has been excreted, low NaCl diet), high urea output (eliminating retained urea) for present low NaCl and low protein diet.

TABLE IX

CHRONIC DIFFUSE NEPHRITIS

Female, unmarried. Age 25. B. P. 148/95.

Blood: Urea N. 12.4 mg. Uric acid 4.0 mg.

Urine: Album. 10 gm. per L. Many hyaline casts. A few R.B.C. Many (x) B.C.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	70	1023				
10–12	80	24				
12– 2	50	30				
2– 4	70	26				
4– 6	70	25				
6–10	150	24				
Total day.....	490	....	1.00	4.90	.7	3.43
10– 8	520	21	.88	4.57	1.1	5.20
	1010			9.47		8.63

Diagnosis: Chronic diffuse nephritis.

Renal Function: Slight impairment (blood uric acid high), albuminuria.

Metabolism: Eliminating retained NaCl and water (diet salt free and fluid intake 800 c.c.).

TABLE X

SECONDARY CONTRACTED KIDNEY

Male. Age 20. B. P. 164/83 to 132/80.
Blood: Sugar .11 per cent. Urea N. 86.2 mg. Uric acid 8.3 mg. Creatinin 5.0 mg.
Urine: Album. 2.5 gm. per L. A few R.B.C. Occasional hyaline casts.
Hemoglobin: 35 per cent. R.B.C. 2,960,000.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	200	1010				
10–12	95	10				
12– 2	100	10				
2– 4	215	09				
4– 6	135	08				
6–10	320	10				
Total day.....	1065	....	.29	3.09	.60	6.39
10– 8	140	10	.40	1.64	.50	2.05
Total.....	1475	....	...	4.73	...	8.44

Diagnosis: Secondary contracted kidney, secondary anemia.
Renal Function: Marked impairment (high blood chemistry, low fixed Sp. Gr.).
Metabolism: No change from therapeutic standpoint possible; the diet is already so low in proteins that further curtailment cannot be considered. Transfusion may be resorted to. Marked retention of urinary excretory products, inability to concentrate urine.

TABLE XI

ESSENTIAL HYPERTENSION

Female, unmarried. Age 53. B. P. 225/125.
Blood: Sugar .12 per cent. Urea N. 12.8 mg. Uric acid 2.9 mg. Creatinin 1.4 mg.
Urine: Albumin 0. Microscopic examination negative.
Hemoglobin: 80 per cent.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	70	1015				
10–12	80	12				
12– 2	80	18				
2– 4	180	10				
4– 6	100	20				
6–10.30	100	25				
Total day.....	610	....	.44	2.68	1.1	6.7
10.30–8	460	15	.49	3.25	1.0	4.6
Total.....	1070	....	...	5.93	...	11.3

Diagnosis: Essential hypertension.
Renal Function: Normal.
Metabolism: Living on a very low food intake, possibly ideal under the circumstances. The urea output in the 24-hour urine is very low; the hemoglobin indicates that at present the food supply is sufficient for maintenance.

TABLE XII

ARTERIOSCLEROTIC KIDNEY (PRIMARY CONTRACTED)

Male. Age 32. B. P. 210/128.
Blood: Urea N. 105 mg. Creatinin 6.7 mg.
Urine: Albumin trace. Few granular casts.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	58	1011				
10–12	90	10				
12– 2	92	11				
2– 4	156	10				
4– 6	86	11				
6– 8	110	11				
Total day.....	592	....	.34	2.01	1.1	6.47
8– 8	1250	10	.49	6.18	.9	11.13
Total.....	1842	....	...	8.19	...	17.60

Diagnosis: Arteriosclerotic (primary contracted) kidney.
Renal Function: Maximal impairment (high blood chemistry, low fixed Sp. Gr., nocturnal polyuria).
Metabolism: Retention of urinary excretory products, inability to concentrate urine. Curtailment of salt and proteins in diet indicated.

EXAMPLES OF TESTS FOR RENAL FUNCTION THAT MAY BE APPLIED TO GENERAL METABOLIC PROBLEMS

TABLE XIII

HIGH SODIUM CHLORID

Female, married. Age 60. B. P. 220/140.
Blood: Sugar .12 per cent. Urea N. 11.2 mg. Uric acid 5.2 mg. Creatinin 1.6 mg.
Urine: Album. ft. tr. No casts. No blood cells.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	174	1007				
10–12	40	15				
12– 2	22					
2– 4	48	25				
4– 7	128	23				
7–10	134	22				
Total day.....	546	....	.76	4.15	1.2	6.5
10– 8	1022	12	.70	7.15	0.6	6.1
Total	1568	....	...	11.30	...	12.6

Diagnosis: Essential hypertension. Slight degree arteriosclerotic kidney.
Renal Function: Uric acid high in blood. Nocturnal polyuria.
Metabolism: NaCl intake high.

This case shows very well how patients who may have been strictly following dietary directions may make some unconscious mistake that has escaped both them and the physician. This individual taking a low protein diet, as shown by the blood urea nitrogen, is indulging in a large amount of salt, which, naturally, is contraindicated in a case where we wish to spare the kidneys as much as possible. The high salt intake and excretion in this instance is probably responsible for the high night urine. Without these tests, the indiscretion in the diet would not have been discovered.

TABLE XIV

LOW FLUID INTAKE

Male. Age 52. B. P. 142/84.
Blood: Sugar .10 per cent. Urea N. 12.8 mg. Uric acid 2.2 mg. Creatinin 1.2 mg.
Urine: Albumin non. Microscopic examination negative.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10.20	85	1030				
10.20–12.40	100	29				
12.40– 2.30	90	28				
2.30– 4.25	70	34				
4.25– 7.00	150	30				
7.00–10.00	170	31				
Total day.....	665	....	1.96	12.03	1.8	11.87
10.00–8.00	280	26	0.70	1.96	1.7	4.76
Total	945			13.99		16.63

Diagnosis: Arteriosclerosis.
Renal Function: Normal.
Metabolism: Low fluid intake. (High fixed Sp. Gr.)

The high specific gravity of this case indicates that the patient is taking too little fluid. The water metabolism is one of the most important functions and it is probably responsible, if neglected, for many cases of constipation. The test here shows distinctly that this individual's urinary specific gravity is constantly above 1026 and is, therefore, much too high. The correction of this fault is easily accomplished.

TABLE XV

LOW PROTEIN INTAKE

Female, married. Age 57. B. P. 128/73.
Blood: Sugar .08 per cent. Urea N. 13.2 mg. Uric acid 1.6 mg. Creatinin 1.1 mg.
Urine: Normal.
Hemoglobin: 77 per cent. R.B.C. 4,500,000.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	80	1019				
10–12	50	24				
12– 2	45	23				
2– 3	360	07				
3– 6	260	10				
6–11	200	15				
Total day.....	995					
11– 8	505	17				
Total.....	1500	....	0.40	6.0	0.55	8.2

Diagnosis: Secondary anemia. Malnutrition.
Renal Function: Normal.
Metabolism: Low protein intake. (The amount of urea put out in the 24-hour specimen indicates that only about 30 gms. of protein are ingested in the course of the day. A higher protein intake would be the rational remedy for the feeling of lassitude, the malnutrition and the secondary anemia.)

In this case the symptom that we have to explain is the secondary anemia. You see that this patient is not retaining the end-products of protein metabolism in the blood, and therefore, the elimination of 8 gms. of urea while she is on her usual diet, would indicate that she is ingesting only about 30 gms. of protein, which is entirely insufficient to maintain life at a proper level. The anemia and malnutrition in this case are evidently due to low food intake, which should be trebled in order to bring about a cure. Nearly all patients become indignant when questioned about their diet, saying that they eat just what every one else indulges in, yet these tests reveal very definitely that far-reaching faults are often present in the dietetic life of many individuals.

TABLE XVI

TOO LITTLE FOOD AND FLUID

Female, married. Age 28. B. P. 126/78.
Blood: Sugar .10 per cent. Urea N. 9.9 mg. Uric acid 2.9 mg.
Urine: Album. tr. Pus cells, a few. R.B.C., occasional.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	40	1021				
10–12	66	18				
12– 2	95	18				
2– 4	58	21				
4– 7	64	20				
7–10	118	17				
Total day.....	441	....	.60	2.65	.64	2.8
10– 8	198	22	.48	0.95	2.22	4.4
Total.....	639			3.60		7.2

Diagnosis: Renal calculus.
Renal Function: Normal.
Metabolism: Fluid, NaCl, and protein intake low.

This case shows a combination of what we have previously seen. The rather high fixed specific gravity, showing that very little water is taken, in this instance becomes of extreme importance. The patient is suffering with renal calculus, and certainly under these circumstances, a large amount of fluid should be ingested. The low food intake possibly here is indicated for the handling of the disease.

TABLE XVII

HIGH FOOD INTAKE

Male. Age 56. B. P. 146/88. Weight 215 lbs.
After low CH diet B. P. 112/72. Weight 191¼ lbs.
Blood: Sugar .16 per cent. Urea N. 11.9 mg. Uric acid 2.8 mg. Creatinin 2.9 mg.
Urine: Album. none. Microscopic examination negative. Sugar none.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	45	1025				
10–12	100	25				
12– 2	140	21				
2– 4	165	22				
4– 6	128	25				
6–10	205	25				
Total day.....	783	....	1.10	8.61	2.3	18.0
10– 8	475	26	.96	4.56	2.7	13.0
Total.....	1258	....	....	13.17	...	31.0

Diagnosis: Obesity; inability to metabolize amount of sugar ingested properly.
Renal Function: Normal.
Metabolism: Too little fluid; excessive starch and protein intake. (Sp. Gr. high and fixed; high blood sugar; excessive amount of urea in the 24-hour urine.)

This patient is extremely interesting from the metabolic point of view. He is a man of good health who is suffering considerably from minor ailments, such as furunculosis, catarrhal condition of the throat and indigestion. He claims that he is a very "small eater." However, we see here that he takes approximately 95 gms. of protein in his food, as judged by the urea output. His starch intake also is high, as shown by his blood sugar, whereas the amount of water he drinks is much too small to keep the urine at a proper specific gravity level in relation to the output of solids. A restriction of the starch diet in this individual and an increase in the amount of water taken proved to be of very great value. His weight dropped, as shown in the table, from 215 to 191 lbs. and at the same time his blood pressure was lowered from 146 to 112. This is not an uncommon experience in obese individuals. This man was eating more than his share of both

protein and starchy food. The high protein portion of the diet did not seem to be doing him any particular amount of harm, and was, therefore, allowed. The starches, however, were having their effect, and had to be curtailed.

The next and last table is an example of the very popular self-imposed diet which many individuals practice today. They are afraid of increasing their blood pressure because of over-indulgence in meats and other highly nitrogenous food and, therefore, eat indiscriminately of starches. In consequence, they become obese and physically below par. What is not generally recognized is the fact that their blood pressure may rise, not in every instance, but in some. In this particular case the blood pressure dropped 40 points after the starch intake was curtailed. This individual, in contrast to the last one, overate only in starch and not in nitrogenous food.

TABLE XVIII

HIGH STARCH, LOW PROTEIN INTAKE

Female, married. Age 64. B. P. 190/100; after diet, 150/90.
Blood: Sugar .246 per cent. Urea N. 19.6 mg. Uric acid 3.6 mg.
Urine: Album. Casts, none.
Blood: After diet, .104 per cent. Uric acid 2.7 mg.

			NaCl		Urea	
Test meal	C.c.	Sp. gr.	Per cent	Gm.	Per cent	Gm.
8–10	29	1021				
10–12	36					
12–2	18	23				
2–4	42					
4–7	50	29				
7–10	78	28				
Total day	253	....	.87	2.21	2.5	6.24
10– 8	545	16	.55	2.98	1.4	7.63
Total	798	....	...	5.19	...	13.87

Diagnosis: Arteriosclerosis, hypertension, myocardial insufficiency, possible mild thyroid insufficiency.
Renal Function: Slight impairment; high blood urea and uric acid.
Metabolism: Marked excess in starch intake; low protein intake; low water intake.

I trust that a demonstration of these methods for testing renal function gives you an idea of how very valuable they

are and how an intelligent treatment of cases of kidney disease is not possible without them. Inasmuch as by these tests we are studying not only renal function, but metabolism as well, it becomes self-evident that we may make corrections in the daily diet and routine of any conditions that will have a far-reaching influence.

It is of the utmost importance to remember that these tests can be carried out while the patients are following their routine occupations and diet with much more value as diagnostic measures than if we have them confined to the hospital under artificial restrictions of food and activity. By carrying out these tests for renal function, and possibly, if you choose to consider them as tests, for metabolism, under routine conditions, we are able to adjust the individual to his environment and really carry out therapeutics much more intelligently than we can under artificially imposed conditions.

EVERYDAY BACTERIOLOGY*

WADE W. OLIVER, M.D.

INTRODUCTION

THE appended notes are addressed to the busy practitoner in the hope that they may contain certain simple practical hints which will enable him to cooperate more efficiently with the bacteriologist. A fact too frequently ignored is that at least one-half the success in bacteriological investigations is spelled in terms of the precision with which specimens are collected and the speed with which the specimens are brought to the laboratory.

SPUTUM

What Is It? Sputum refers to the secretion from the lower respiratory passages and it is obtained by a deep pulmonary cough. Sputum is not saliva which dribbles from the mouth, nor is it nasopharyngeal secretion.

How Obtained? By having the patient indulge in a deep pulmonary cough after he has gargled the back of his throat three times with sterile water to clean his mouth.

Where Collected? Have the patient void the sputum into a sterile container, preferably a wide-mouthed bottle. (Caution him against soiling the outside of the bottle with sputum.)

Precautions. 1. Keep the container tightly corked, except during actual collection of the material. This is to avoid gross contamination from the outside.

2. Label the container plainly with patient's name, nature of specimen and object of examination. The bacteriologist is no soothsayer. You may know what you want the specimen examined for, but the bacteriologist will not unless you tell him.

* This lecture was delivered on April 13, 1923.

3. No disinfectant of any kind should be added to the sputum. You cannot expect the pneumococcus to grow if it is sent to the laboratory pickled in 1 per cent carbolic acid.

4. Send, or better, bring the specimen to the laboratory as soon as it is obtained.

Throat Cultures

Collection. Usually throat cultures are taken by swabbing, with a sterile cotton swab (mounted on a sterile stick), the inflamed throat area.

In the case of diphtheria, a superficial touching of the membrane with the swab may fail to yield B. diphtherial when the swab is streaked over a slant of Loeffler's blood serum. When possible, get a piece of the membrane or make the swab between the diphtheritic membrane and mucous membrane.

Precautions. 1. Never use an old dried-up slant of Loeffler's blood serum.

2. If delay is necessary in getting the inoculated tube to the laboratory, incubate the tube in your vest pocket while it is in transit.

3. Always make a smear from the membrane direct. Fix it by passing it rather rapidly through the Bunsen flame three times and bring the smear to the laboratory along with the inoculated Loeffler's tube. In at least 75 per cent of the cases of diphtheria, a diagnosis can be made from the direct smear from the membrane. This means twelve to sixteen hours saved in the administration of antitoxin.

4. If the case be not diphtheria, but Vincent's angina, the smear is absolutely necessary, because the diagnosis of Vincent's angina is made by finding the fusiform bacillus and spirochete in large numbers in direct smears from the throat.

Blood Culture

What Is It? It is the endeavor to grow bacteria from the circulating blood. A positive blood culture is always of pathologic import, because normally the blood is sterile.

Apparatus Required. (*a*) Syringe. An all-glass Luer syringe of 10 c.c. capacity with a platinum iridium needle of moderate bore. The syringe is best sterilized, taken

completely apart, in the autoclave enclosed in a large plugged test-tube, at 20 lbs. pressure for 20 minutes. Such a syringe will be "bacteriologically sterile." A syringe is rendered "surgically sterile" by boiling in water for 10 to 30 minutes, but "A syringe cannot be sterilized for bacterial purposes by boiling in water." *Kendall.*

(*b*) Sterile test-tube containing about 1 c.c. of a 2 per cent solution of sodium citrate or 1 per cent ammonium oxalate; or a test-tube containing several sterile defibrinating sticks or glass beads may be used.

(*c*) Sterile gauze, tincture of green soap, alcohol and tincture of iodine.

(*d*) Tourniquet: a piece of medium-soft rubber tubing.

Collection of Blood. (*a*) Site of choice: area around median basilic vein, at inner aspect of forearm. Cleanse area first with tincture of green soap, followed by alcohol. If veins are prominent, next apply tincture of iodine.

(*b*) Apply tourniquet to arm above the elbow with moderate pressure, to impede venous flow, and cause the vein to become prominent. Have patient make and open fist several times if vein is difficultly visible.

(*c*) Insert needle gently into the vein, from below up parallel to the vessel and at an angle very acute to the surface. The beveled part of the needle should be up. Withdraw 5 to 10 c.c. of blood.

(*d*) Loosen the tourniquet and withdraw needle from vein.

(*e*) Remove the needle carefully from the syringe and squirt the blood immediately into the test-tube containing sodium citrate. Hold the test-tube in a slanting position and flame the mouth of the test-tube after removing the cotton plug and upon replacing the cotton stopper.

(*f*) Admix the blood and citrate by gently shaking and rotating the tube. Avoid wetting the cotton plug with blood. Cotton is an efficient bacterial filter only so long as it is dry.

(*g*) Bring blood to the laboratory immediately.

Precautions. 1. In drawing the blood, avoid entrance of air into the vein by avoiding pressure on the plunger and by not allowing the plunger to slip back.

2. Look upon the taking of a blood culture as a surgical operation, during which all the rules of sterilization and asepsis must be observed.

Why? (*a*) For the patient's welfare and safety.

(*b*) For the bacteriologist's welfare and temper.

Improperly collected blood will be contaminated, in which case the blood culture made by the bacteriologist will be contaminated, and the results may be entirely worthless.

Addendum. If a Widal test for typhoid is desired, squirt the blood into a dry sterile test-tube and allow it to clot in a slanting position. This makes it easier for the bacteriologist to collect the serum.

Typhoid Blood Cultures:

224 cases during first week of disease—89 per cent positive.

484 cases during second week of disease—73 per cent positive.

268 cases during third week of disease—60 per cent positive.

103 cases during fourth week of disease—38 per cent positive.

58 cases after fourth week of disease—26 per cent positive.

Feces

Approximately 8 gms. of bacteria weighed dry are passed per day by a normal adult, when the total weight of the dry feces is 27 gms. In other words, nearly one-third of the weight of dry feces represents bacterial cell bodies. It has been well said: "The alimentary canal is probably the most efficient combined culture medium and incubator known to bacteriology."

Collection. For bacteriological examination, the stool is best sent to the laboratory in a small wide-mouthed bottle, tightly stoppered with a cork stopper, into which fits a stick flared at the free end. On the flat end of the stick, a portion of stool about the size of the little finger nail is collected, the material is carefully placed in the container and the cork tightened.

If the stool is fluid, 1 to 2 c.c. of stool can be carefully poured into the container, which must be tightly corked.

Remember. It is only necessary to send the whole stool when the feces are to be examined for animal parasites. Then the specimen should be obtained after a purge.

Also Remember. In order to find the typhoid bacillus, which is about $\frac{3}{25,000}$ of an inch in length, it is not necessary to send a gallon of feces to the laboratory.

Precautions. 1. Never use cardboard boxes as stool containers, especially if the stool be fluid in nature. Any container which may leak should never be employed.

2. Always make sure the feces container is tightly stoppered.

3. Do not smear the mouth of the container and cork with feces.

The above precautions are especially imperative in the case of stools sent from patients suffering from such diseases as typhoid, paratyphoid, and dysentery. Failure to observe these precautions by a doctor, nurse or orderly is nothing short of criminal, because the three "F's" of bacteriology (fingers, flies and food) are the usual ways in which such diseases are spread, and any carelessness in the disposition of feces may result in spreading the infection.

Urine

Normal urine is sterile.

Collection. 1. In men: It is seldom necessary to use the catheter to obtain a specimen for bacteriological examination. Cleanse the glans and meatus thoroughly with tincture of green soap and water. Collect only the last 10 c.c. of urine, which will be relatively free from extraneous microorganisms, due to the mechanical flushing action of urination. Collect the specimen in a wide-mouthed sterile bottle, tightly stoppered.

2. In women: A satisfactory specimen can be obtained only by catheterization after preliminary cleansing with soap and water.

Common Pathogens. Staphylococcus, Streptococcus, B. coli, Gonococcus, B. typhosus and B. paratyphosus, B. proteus, B. tuberculosis.

Spinal Fluid

Normal spinal fluid is sterile.

Collection. The fluid should be collected in a sterile test-tube and sent *immediately* to the laboratory. A delicate microorganism such as the meningococcus dies in four to six hours if the spinal fluid stands at room temperature for that period of time after withdrawal.

Common Pathogens in Spinal Fluid. Fluid clear. B. tuberculosis, Treponema pallidum, virus of poliomyelitis.

Fluid Cloudy. Meningococcus, Streptococcus, pneumococcus, B. influenzae, B. coli, B. typhosus, B. pyocyaneus.

Direct Smears for Acid Fasts. Tuberculous spinal fluid usually is clear. Upon standing, however, cobweb-like coagula appear, in which are enmeshed bacteria and cells. In about 75 per cent of cases of tuberculous meningitis, direct smears of the coagula will reveal the presence of acid-fasts.

Vaccines

Definition. A vaccine is a killed or weakened culture of the bacteria with which the patient is infected.

An autogenous vaccine should always be used whenever possible. By an autogenous vaccine is meant one in which the organism used was isolated from the particular patient who is to receive the vaccine.

A stock vaccine is one in which the organism was isolated from some patient suffering from a particular infection, such as furunculosis, and the strain is kept going in the laboratory, and from this "stock" strain vaccines are prepared.

Reasons for the Superiority of an Autogenous Vaccine Over a Stock Vaccine. 1. Any given bacterium may change in important chemical properties on being grown for a few generations outside the body on artificial culture media. A vaccine should preferably be prepared from a culture of bacteria not more than two generations removed from the patient in which they have been doing mischief.

2. Most, if not all, disease-producing bacteria represent a large number of more or less physicochemically distinct strains. If an autogenous vaccine has been properly prepared, you can be sure you are injecting the particular strain of

bacterium into your patient which was responsible for his lesion. If you employ a stock vaccine, you are not sure whether that stock vaccine contains the particular strain of organism or not.

3. The stock vaccines for furuncles contain Staphylococci. Certain cases of furunculosis are not Staphylococcus infections at all, but may be due to an entirely different organism, such as B. coli.

The ordinary vaccines employed in general practice consist of salt solution suspensions of bacteria which have been killed by heat. This is true of such vaccines as typhoid vaccine, coli vaccine, Staphylococcus vaccine, etc. Smallpox vaccine, however, consists of the living virus of smallpox which has been weakened by passage through the body of a calf.

Principle of vaccine action: The rationale of vaccine therapy briefly is this—if you inject into a healthy tissue of the body, first, minute, and then gradually increasing doses of killed or weakened bacteria of the particular kind responsible for the patient's infection, the healthy tissues of the patient's body are stimulated to produce specific bacteria destroying substances (antibodies) which contribute to the destruction of the offending living bacteria in the diseased area.

Vaccines set up an active immunity, i.e., the patient's body is the active agent in the production of protective antibodies.

Collection of Pus for Preparation of an Autogenous Vaccine

From Boil. (a) Cleanse skin around and over boil thoroughly with tincture of green soap and water, followed by 50 per cent alcohol. (Tincture of iodine acting for three minutes may be applied following the alcohol.)

(b) Carefully puncture the boil with a sterile needle. Squeeze lightly around base of boil if necessary, to express pus.

(c) Carefully collect pus on a sterile cotton swab mounted on a sterile stick. Replace swab in a sterile test-tube and

bring it immediately to the laboratory for plating. From such material an autogenous vaccine is prepared.

Serums

Types. 1. Normal. 2. Immune.

1. Normal serum is the clear straw-colored fluid expressed from the blood of a normal, untreated animal during the act of clotting.

2. Immune serum is serum obtained from a naturally or artificially immunized animal. Such a serum, depending upon the nature of its production, contains protective substances known as antibodies.

Examples. (a) Anti-diphtheritic serum is serum obtained from a horse which has been injected over a period of months with gradually increasing doses of diphtheria toxin. Such injections of toxin stimulate the body cells of the horse to the production of antitoxin, an antibody which possesses the specific power of neutralizing, or rendering inert, diphtheria toxin. Serum therapy in diphtheria is employed as early as possible in the course of the disease, in order that the circulating toxin produced by the diphtheria bacilli multiplying locally in the throat may be neutralized by the injected antitoxin while the toxin is still circulating free in the blood stream.

(b) Antipneumococcus serum (Type 1) contains antibodies which lead to the destruction of Type 1 pneumococci.

Remember. Serum therapy sets up a passive transient immunity.

Vaccine therapy set up an active, more lasting immunity.

In serum therapy preformed protective antibodies are injected, and they tend to be rather quickly excreted in the urine, sweat, etc.

In vaccine therapy the vaccinated individual is stimulated to produce his own protective antibodies, which persist in his body for a longer period of time.

DRY LABOR*

JOHN OSBORN POLAK, M.D.

WHEN your committee asked me to discuss some obstetric problems, I decided on dry labor and breech delivery, two propositions which give the practitioner considerable trouble.

Some of my statements may sound dogmatic, but they are based on long experience, checked up by results; and it is results that count. No problems in any branch of medicine are so little understood as are the obstetric problems.

In the first place, dry labor, as you know, is a most serious complication, not only from the standpoint of the mother, but from that of the child; and it occurs with relative frequency. In dry labor we have a condition which permits infection, not only from the intrapelvic manipulation that may be necessary, but from the fact that the membranes are ruptured. This deprives the fetus, the placenta and the placental site of their former protection by intact membranes. The absence of the protecting bag of waters allows the bacterial flora of the vagina to migrate into the uterus, and the longer the patient is in active labor and the presenting part pounds against the unopened cervix, the less is the tissue resistance. For it has been demonstrated that the amnion, one of the coverings of the baby, covered as it is, with protective cuboidal cells, loses its resistance, the cells become edematous, and the position of the nuclei in the cells changes as the labor goes on. This destroys one of nature's protections and permits penetration of the amnion by bacteria. This point has been proved both clinically and bacteriologically by a number of excellent observers.

From the obstetric standpoint in dry labor, *it is the cervix which offers obstruction;* and by offering obstruction it excites a reflex irritability in the uterus and we find in the uterus the formation of a retraction ring. When this phe-

* This lecture was delivered on Oct. 20, 1922.

nomenon occurs, it so moulds the uterus on the body of the child that it interferes with the uteroplacental circulation, and so the child's circulation, or, as Crile says, its internal respiration, is interfered with, and it is frequently born dead. Again, in a large number of cases of dry labor the baby is born alive, but dies within a few days, and at autopsy cerebral hemorrhages are found. This is because the child's head has pounded hour after hour, and as a result of placental compression, cerebral congestion takes place and the delicate capillaries in the brain are ruptured. This produces a wet brain (cerebral hemorrhage), from which the child dies four, five or six days afterwards.

From the standpoint of the mother, dry labor is serious. It is exhausting, for not only is the possibility of infection greater, but the woman is more likely to sustain lacerations and injuries. The likelihood of operative interference is also greater. Furthermore, the tired uterus is more likely to bleed—hence blood loss from post-partum hemorrhage is not infrequent. This further lowers the patient's resistance.

I shall attempt to explain what the presence of intact membranes means and discuss the changes that take place in the cervix and uterus when the dilating bag is present. I shall also suggest substitutes for this dilating fluid wedge, when the waters have drained away.

Dry labor may be considered under three general heads:

The first, when the membranes have ruptured before labor pains have occurred, with the head in the brim and the cervix not dilated, but where the size of the child and the size of the pelvis are normal or relatively normal.

The second group for consideration includes those cases after labor has begun. The membranes are ruptured; with the head or the breech in the pelvis, the cervix is uneffaced and undilated, but the woman is having active labor pains.

In the third class are included the cases in which the membranes have ruptured, but the head or breech has not engaged though the child and the pelvis are relatively normal; and where, if the membranes were intact, you and I would give the woman a full test of labor; but with the membranes absent we know that the child will suffer as the result of prolonged, repeated contractions.

Recently we reviewed the histories of 1700 labors occurring in our clinic at the Long Island College Hospital, in order that we might determine the incidence of dry labor, the frequency of spontaneous birth and the incidence of operative intervention. No labor was classed as prolonged unless the membranes had been ruptured for over twenty-four hours. In these 1700 labors we had 266 dry labors.

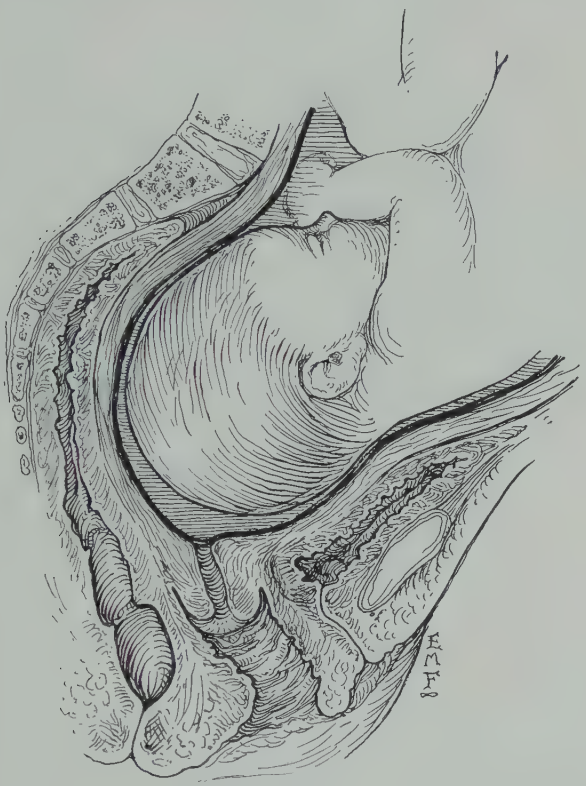


Fig. 101. Unprepared cervix before lightening has taken place.

Very few practitioners have a real conception of how the cervix opens; and, of course, it is necessary for the cervix to be open before the baby can come out. Figure 101 shows a primiparous cervix at the end of pregnancy, before labor has taken place, while Figure 102 represents the same cervix after labor has just begun. What change do we notice? Observe

that the cervix is shortened; that is, the canal is shortened, and the internal os shows some obliteration.

In Figure 103 the internal os has disappeared, but we have an external os; and it is not until the cervix has been prepared by the effacement of the portio and obliteration of the internal os, that dilatation or opening of the cervix actually

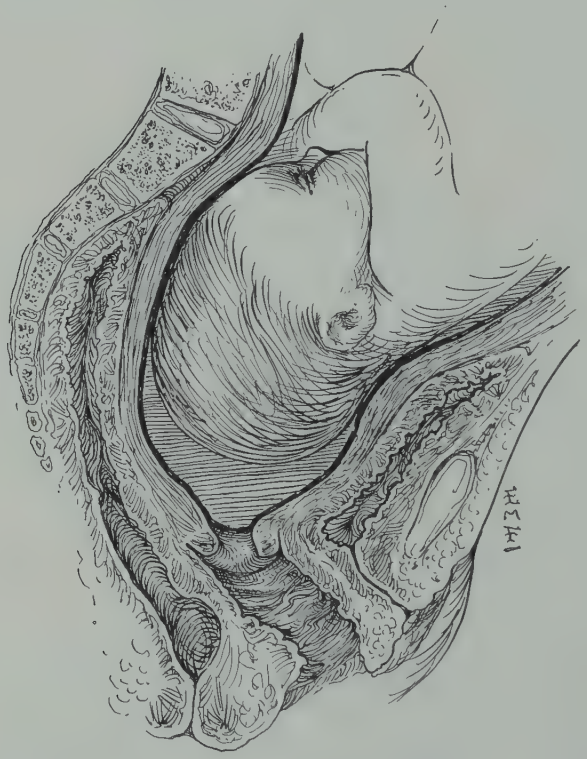


FIG. 102. Showing shortening of the portio and obliteration of the internal os.

takes place. The cervix opens as a result of uterine contraction which pushes the bag of waters through as a hydrostatic bag. Or if the membranes are ruptured, the presenting part with its caput is driven into the os.

There is a difference between the multiparous and the primiparous cervix. In the multipara the cervix is more open before labor begins. There is not the degree of obliteration

of the internal os and not quite as much shortening of the cervical canal, and when the dilatation of the cervix is complete, we still have a rim of cervix which is not absolutely obliterated. The primipara with perfect dilatation has nothing left of the cervical rim; the uterus and vagina become one continuous passage. In the multipara, on the other hand, there is a cervical rim about the vaginal circle.

Fig. 103. Internal os completely obliterated; external os dilating.

Another fundamental principle that should be emphasized is that the cervix must be completely dilated before we can successfully deliver by either forceps or breech extraction. We all do a great many forceps deliveries, and they will present no difficulty if we wait for full dilatation and for the head to reach the spines. You have perhaps heard of a man in Buffalo who turns every case, doing a version in order that the woman may be saved a couple of hours of pain in the

second stage of labor, but he waits for full cervical dilatation; and the one thing we are trying to teach is that there will be no trouble with forceps or extraction after version if one will have patience and not interfere in the presence of an incompletely dilated cervix.

All labors may be divided into two classes: those cases in which the presenting part will come into the pelvis and those in which it will not. There are no "ifs" or "ands." A head will come in or it will not come in. If it comes in, it will become flexed and definitely engaged. If it does not, it remains as a floating head. A floating head—one that does not enter the brim and engage—will not admit of intrapelvic delivery. When a head is engaged, the greatest diameter of the greatest circumference has entered the brim; the greatest circumference has passed through the pelvic brim. When the head is really engaged, the vertex is at the spines. No matter what type of forceps you own, you can't deliver with them unless the head has passed the brim and is in the pelvic cavity.

To understand what is meant by "engagement of the head" seems to present great difficulties for some practitioners. When the head is engaged, it means that the greatest diameter of the engaging circumference has passed through the brim of the pelvis. If you wait for this to occur, you won't have difficult forceps cases, because your cases will divide themselves into those that will and those that will not come in, and the result is you will never have anything more difficult than a median or low forceps. You will never have to use high forceps, because the head that is not properly flexed will not come in and cannot be brought through.

Where there is disproportion between the head and the pelvis, the head will not come into the pelvis, even after the cervix is fully dilated.

Figure 104 shows how the internal os is practically obliterated and how the lower segment is thinning out, while the portial cervix is shortened. This is the reason why the cervix opens. The membranes separate from the lower uterine segment, and this allows them to act as a fluid wedge. In the interval between pains the separated membranes retract.

During the pains the fluid bag with its excentric pressure is pushed down and protrudes through the cervix and effects, first, the obliteration of the internal os and then the dilatation of the external os.

I am calling your attention to these basic facts because it has been said by some of my friends that they can by bags or manual dilatation simulate nature. I tell you you cannot,

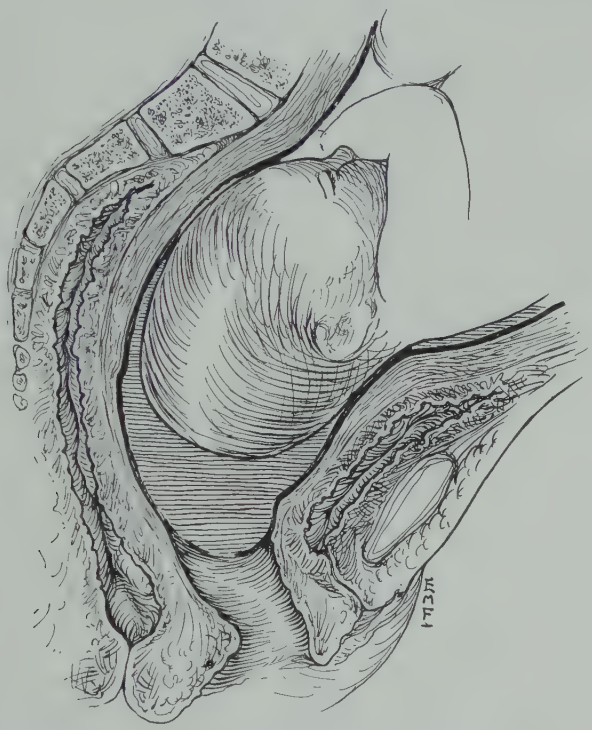


FIG. 104. The engaged head. Cervix fully dilated; membrane intact.

because the mechanism is absolutely perfect; it is gradual and progresssive until the cervical rim is obliterated and paralyzed.

As labor advances, the bag of waters is protruded through the cervix. The posterior lip recedes and the bag of waters is pushed into the vagina and dilates it, just as it has dilated the cervix by intermittent fluid pressure. This is the reason

why midwives who are taught to keep their hands out of the vagina, and to retain the membranes, can frequently deliver a woman with less laceration than you or I can. It is because they do not rupture the membranes until they show outside the vulva. The bag of waters has dilated the passages. The uterus is carried up. The posterior lip recedes first and the anterior lip, which is the lowermost, follows. This accounts for the fact that the cervical tears which always occur in dry labor show the anterior lip to be thicker and longer.

I am often asked the question: "When shall I rupture the membranes?" I reply by stating that if labor is advancing properly, do not rupture the membranes until they are at the vulva, because when you have dilatation the second stage follows rapidly, and the soft parts have been prepared by an elastic pressure. Hence there is less trauma to the vaginal soft parts.

It has been claimed that if the membranes are ruptured, it is an easy matter to dilate the cervix. Certain methods have been suggested. First, is the use of the bag. The bag has certain disadvantages which I shall discuss in detail in a few moments.

Then there is manual dilatation. I should always prefer to call this *manual laceration*, for it is impossible to simulate nature's methods with the hand.

My friend Professor Peterson, of the University of Michigan, said to me one day: "What is the use of teaching our students to dilate the cervix manually? It would be better for them to cut the cervix and get a clean wound which will heal, than to attempt manual dilatation and get a lacerated wound. I am teaching my boys to cut the cervix." But this takes some training and is not a procedure for the beginner.

In addition to the hydrostatic bag and manual dilatation, we must consider the vaginal pack and Duhrsen's multiple incisions.

What interests us in this discussion of dry labor is: When should we use these means of dilatation? When should we not use them?

In dry labors we have learned to study the action of the uterus and note what the pains are actually accomplishing. Is the cervix becoming effaced? Is the os thinner and more

dilated? Is the uterus relaxed between pains? A woman who starts in labor with the membranes ruptured is apt to have irregular, short, cramp-like, nagging, but powerless contractions. She is often more or less exhausted by the time she gets a dilatation of 5 cm., while the unsupported cervix becomes edematous and thickened, and this in turn increases the irritability of the uterus. Unless such a patient is given sleep, rest and food, the inertia will continue. But empty the bladder and lower bowel and give her some food and a quarter of a grain of morphin and she will go to sleep. When she awakes, her pains will be strong and regular, and after her morphin the cervix almost melts away.

In what cases can we use morphin, and in what cases should it not be used? If the head is engaged, or engaging, and the cervix thinned out and dilated, 1 or 2 cm. morphin and scopolamin will give the patient great comfort and expedite the dilatation. We can also use it tentatively in some of the cases of thickened cervices with irregular uterine contractions, as referred to above; but, as a rule, we will find that in cases where the cervix is actually thickened by edema, a pack will be a valuable adjunct to the morphin.

In dry labor I do not use morphin except during the first stage, unless the woman has a thinned-out cervix; then I give morphin in good doses because I know the cervix will simply melt away as the woman goes along.

What are the disadvantages of manual dilatation? In the first place, the interal os must be obliterated and there must be nothing left but the external os, before the cervix can be manually dilated. Again, we cannot with the hand make equilateral pressure in all directions. The result of this is that when we get to the point where five fingers will pass through the cervix, if the patient is well under an anesthetic, the cervical ring suddenly gives way and the hand goes through; and after labor, when we expose the cervix we find an extensive laceration going up on either side into the fornices.

The two-hand method of dilating the cervix was suggested years ago by my distinguished friend, Dr. Edgar, who employed it when the head was deep in the pelvis and the internal os obliterated. The index finger of each hand is passed through the external os and rotary excentric pressure

made; then two fingers of each hand are inserted and the divulsion is continued. This method has been modified by Wetherhill, of Denver, who takes two Sims' specula and uses them instead of the fingers for dilating the cervix, by rotating them within the cervical ring, ironing out the tissues. It is applicable only in cases where the head is low and where the cervix is almost at the vaginal orifice; *but it always ends*

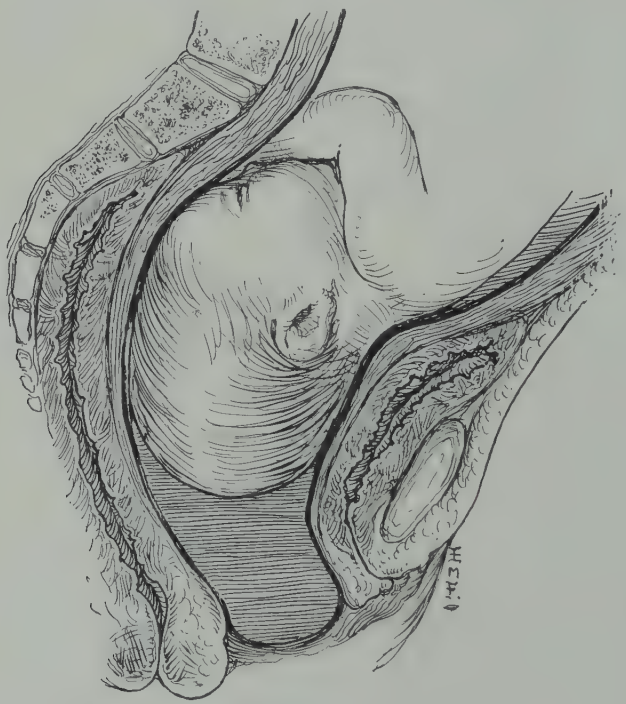


Fig. 105. Cervix fully dilated. Intact membranes pushing down into the vagina, dilating it.

in the same thing, namely, some degree of lacertion. Needless to say, I do not approve of these methods. It is true that every woman is lacerated, but not to any great degree; and in our experience, the more morphin we use and the less manual aid we give, the better it is for the woman.

The hydrostatic bag has many enthusiastic advocates, and in some clinics it is considered as the panacea for dry labor. I want to state what the bag does. If we use a bag

sufficiently large to do any good, it displaces the head. The moment we do this, the head becomes deflexed. It has taken a long time for the head to come into the pelvis and mould itself to the pelvis, yet by the introduction of the bag we

Fig. 106. The hydrostatic bag. It displaces the presenting part, deflexes the head and creates a retraction ring.

push it out and put it on top of the bag, which deflexes it. It also creates a second retraction ring above the edge of the bag, and when the bag is expelled and the cervix is sufficiently open to let it out, we are amazed to find that the head is just above this retraction ring.

Dr. Dorman, of New York, for many years a most ardent advocate for the use of the bag, recently made the statement that 54 per cent of his bag cases ended in operative delivery. If I had such results as these I would certainly rather do a Potter version under full anesthesia.

Furthermore, when the bag is expelled, it is not uncommon for some other parts to come down, for the presenting part is displaced and consequently doesn't block the brim. Personally I have had several complex presentations—a head and a hand, and prolapse of the cord—for by introducing the bag we make a floating head of one that was corking the brim. The bag also serves to dam up the secretions behind it, and as a result parametritis is a very common sequel.

The vaginal bag suggested by Braun, of Vienna, has considerable practical value, for it does not displace the head out of the pelvis. It is placed in the vagina, and in dry labor where a caput is formed on the head, the bag, which tightly fills the vagina, compresses the cervix up against the head and thus shortens the cervix by pressure. The upward pressure of the bag does not allow the cervix to hang down and become edematous and thickened, and as a result of the combination of morphin and the bag, the cervix seems to melt away with comparative rapidity.

The vaginal bag is safe and efficient. It must be a boiled bag and passed through a clean vulva; otherwise its use is dangerous.

For nearly thirty years we have employed aseptic plugging of the vagina with satisfactory results.

As I have already stated, we divide dry labor into the cases where the waters rupture *before labor takes place.* You say: "What shall we do?" Leave the woman alone; don't do anything except keep her in bed. Also keep your hands out of the vagina, so as not to infect her. The woman is more liable to infection after the membranes are ruptured, and the waters won't all leak away, because the head acts as a ball valve and retains a certain amount of hind waters until she starts in labor. Again, you ask: "How long is it safe to wait for labor to set in?" I have had women run along for two weeks after the membranes were ruptured without going into labor and without my subjecting them

to any vaginal examination, and there was delivery of a living child spontaneously when they finally went into labor. You might ask me: "How many babies are lost by following such a 'stand-pat' method?" I have lost one baby in ten years in this class of cases. This woman went for twenty days. As I look back on this case, considering the age of the patient, I should have sectioned her, for this was her only

FIG. 107. The vaginal bag—supporting the cervix and compressing it against the advancing head.

child and she had had an amputation of the cervix for chronic cervicitis. She was thirty-three years of age at the time and had been married ten years before becoming pregnant. Today with such a history we would all say section. I advise section only in those cases where the child becomes of great legal importance and you acquaint the mother and the father with the fact that the operation of cesarean is not free from danger.

In the cases where the membranes are ruptured and the woman is already in labor, if the cervix is thinned out and she is having good, strong, regular pains, there is only one thing we need; that is to give her a dose of morphin and scopolamin and time, and she will come through. On the other hand, if the cervix is rigid and thick we must prevent

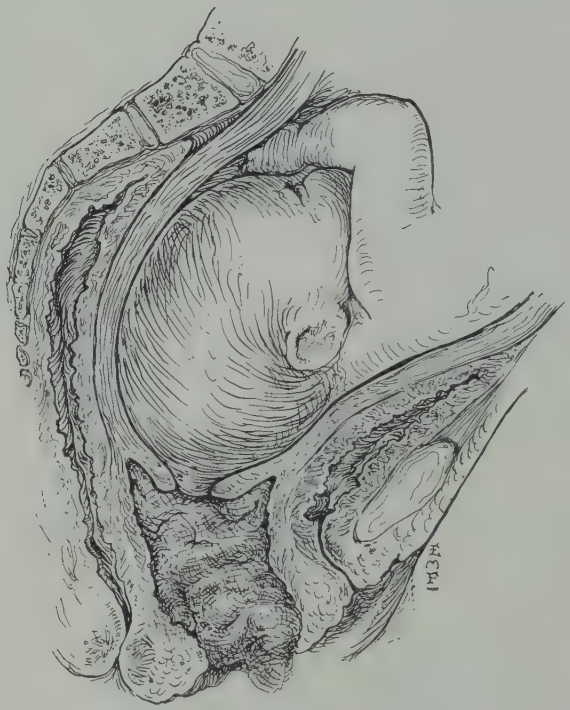


Fig. 108. The vaginal pack, properly placed, helps to soften and dilate the cervix.

further edema and thickening of the cervix, for this interferes with dilatation. This can be done by placing a bag in the vagina underneath the cervix, or by packing the vagina with sterile gauze; or, better still, with boiled cotton batting which has been soaked in sterile boroglycerid, 1 to 8, packing the vagina as firmly as possible, with the patient in the Sims position, and then giving her a dose of morphin. What is the result? In three or four hours the vaginal pack is

expelled, and the head, instead of being retained by the cervix, is in the vagina where it begins its pelvic floor mechanism, having passed through the cervix. Years ago the late Dr. Bodkin taught me this procedure. We also get a hydroscopic action from the glycerin, with a reduction of the edema of the cervix, which makes it more dilatable. At the same time we have the cervix compressed between the advancing head and a firmly placed vaginal pack. In many cases the cervix melts away. Absolute asepsis is necessary. The hair must be cut away from the vulva, though it is not essential that the vulva be shaved, and the vulva scrubbed, using cotton and soap (ivory soap will do as well as any). After that, the soap suds are removed, and for general practice lysol is as satisfactory an antiseptic as any. After the vulva is thoroughly cleansed, we take a Sims speculum, a pair of dressing forceps and a pair of gloves and boil them. They must be sterile when we use them to enter the vagina, but for rectal examination this is not necessary. In dry labor the progress should be followed by rectal touch—not by repeated vaginal examinations, which only contaminate the birth canal. We can follow the progress of dry labor hour by hour by rectal examinations, noting the size of the cervix and the descent and position of the presenting part, and watching the condition of the child by auscultation of the fetal heart with the De Lee stethoscope. This is an accurate and most valuable instrument, because with it we can always hear the fetal heart beat, if there, as it is transmitted like the sound of a tuning fork through the bones of the head to the auditory apparatus.

The fetal heart should be constantly watched after the head has entered the cavity of the pelvis, and particularly after it has passed into the vagina; for after the escape of the head from the cervix, the uterus contracts down upon the body and the uteroplacental circulation is interfered with.

We next come to the consideration of that class of cases where the head is not engaged, or is just engaging, and the cervix admits but one or two fingers; where, ordinarily, you and I would give the woman a test of labor, if the membranes were intact. The question is how to handle this type of case. It very often needs some surgery. Ordinarily, if the mem-

branes were unruptured, we would give the woman a full test of labor; and in 82 per cent of cases she would drive through. But the membranes not being present, the undilated cervix offers resistance, and we have a soft part dystocia added to a slight disproportion. The question is: How long should we leave her? If she has had active labor for twelve hours or so and shows no advance of the head into the pelvis, the child's chances are diminishing hour by hour. The woman's chances are also diminishing hour by hour; for if you contemplate doing any operative procedure, such as a version or cesarean section, you are doing it on a patient who is potentially infected.

The longer you wait, the less are the patient's chances; and let me warn you that in doing cesarean section there are three things which contribute to mortality and to morbidity:

First, the length of time the patient has been in labor.

Second, the length of time the membranes have been ruptured.

Third, the number of times you have examined the woman vaginally.

These statements are based upon an analysis of 2200 cases of cesarean section which were carefully gone over. This study shows that the mortality from cesarean section varies between 3 and 14 per cent, depending on whether the operation was done early in labor, whether the membranes were ruptured and also the number of vaginal examinations. The longer in labor, the longer the membranes have been ruptured, and the more vaginal examinations the patient has had the more certainty is there of infection following cesarean section.

Where we have a case with an unengaged head in dry labor, such a case must be treated with the idea in mind that it may be a borderline case, and if so, operative intervention may be necessary. Therefore, watch the patient expectantly for at least twelve or fifteen hours. Don't make vaginal examinations. Don't let her go too long, or she will be exhausted; and exhaustion favors post-partum hemorrhage and infection.

Therefore, in closing, I want to say that cases of dry labor *should not be bagged.* We should remember that these cervices

ordinarily dilate normally with the bag of waters; that there is no substitute for the bag of waters; that the more closely the cervix is applied to the head, the less edema in the cervix and the less thickening, and the more easily the cervix melts. That is the reason why the vaginal bag and the vaginal pack, in the cases in which the membranes are ruptured, are always combined with morphin so that the patient may rest between her pains; and don't forget that a rigid cervix causes a spastic uterus.

QUESTIONS AND ANSWERS

Q. What about the effect of morphin on the child?

A. Unless you are going to do a cesarean section, morphin has no effect on the child. I will cite certain instances to prove this point. A very interesting case was reported recently by Dr. Brown of Detroit, of a morphin habitué who was taking 14 grains of morphin a day. She was pregnant, and the baby naturally got used to morphin, as you can readily see; but now the lesson: She had 8 grains during her six-hour labor and the baby cried lustily at birth. The pediatricians said: "This baby cannot live on a morphin milk, so we will give it artificial feeding," which they did. After being on the formula, the baby did not thrive. Finally another pediatrician came along and said: "We will have to get some of this milk," and started to give the baby a little breast milk. As soon as it took the milk, it became comatose and died with all the symptoms of morphin poisoning. Morphin was not passed through the placental circulation to the baby, but it was passed through the milk.

Now, in eclampsia every one of us has given morphin. I know of one case where one of my residents gave 5 grains of morphin to a patient in the course of a forty-eight hours' labor. Yet the baby was born spontaneously and lived. I have observed, however, that where morphin has been given prior to cesarean section, the baby is slow to respire.

Within four hours of delivery we may use morphin in ¼ or ⅜, or even ½ grain doses, if necessary, during the first stage of a long labor with perfect safety to the baby; *but not in the second stage.*

Q. In the use of the vaginal bag do you always wait for spontaneous expulsion?

A. Personally, I think it is better to wait for spontaneous expulsion unless the baby's heart shows signs of failure. We would then have to do something more radical. I have not seen one of these cases yet, but it may happen. If we wait for spontaneous expulsion we know that the presenting part is close behind.

Q. Do you ever use pituitrin to assist the pains?

A. Pituitrin in dry labor? Never. There is only one time when pituitrin is absolutely safe, and that is after the baby and the placenta are out. With a cervix rigid and edematous and already causing reflex excitability in the uterus, if we give any amount of pituitrin, we will have a spastic condition that will never get the baby out, and it will in most instances die. I have killed three babies myself, and I have reason to think they died following my dose of pituitrin. In one case I had to give the woman chloroform for a half-hour until the effect of the pituitrin had worn off. It caused the baby's heart to drop from 130 down to 6 per minute, during the spasm of the uterus; and it was not until I pushed her with chloroform and relaxed the uterus that the circulation was reestablished.

Q. How do you handle the patient's relatives and friends?

A. That is a most excellent question. I do not know of anything more important. I practiced $10.00 and $15.00 obstetrics for twenty years among the same people with whom you are now working. There is a psychology in handling the family. I cannot tell you how to do it, but I can do it for you.

The point is this: be absolutely sure of yourself. Don't talk too much. Show them that you are the master of the situation from the minute you get there, and you won't have trouble with the people. You may say that this is all imagination, but many men know perfectly well that we can handle people in that way. You must not let a woman cry out and suffer. If she has many people around her, get rid of most of them. The fewer she has around her the less she will cry.

Q. Do you ever repeat the morphin?

A. We do, in ⅛ grain doses in the first stage, if the labor is prolonged.

THE MANAGEMENT OF THE BREECH*

JOHN OSBORN POLAK, M.D.

THE first part of this paper will deal with the subject of breech presentation. Later, I will attempt to demonstrate the so-called "Potter version" which has had so much criticism. The Potter version is nothing more or less than a collection of all the good points of technique in version which have been taught for years, correlated by Dr. Potter into a working whole. While I am not in accord with Potter as to his indications for version, I am willing to admit that he is practicing today the best technical version. This does not mean, understand me, that we should interfere on the indications upon which he interferes, for from my study of his results, I do not feel that his interference is justified. I will compare his results with results in 1000 cases taken from our service at the Long Island College Hospital, where prenatal work was done. Here the women were confined by an intern or a resident, not by an expert. Potter's fetal mortality is three times greater than ours, yet he has done over 900 versions and 80 cesarean sections. Certainly such figures must convince you that nature is competent, in the vast majority of instances, to do her own obstetrics, if you know the diagnosis and are dealing with a head, pelvis and uterus that are relatively normal.

The next point that I wish to emphasize is that the ordinary breech case, as you and I see it, will deliver itself aided by what I shall call *intelligent aseptic expectancy*. That is, keeping your hands clean, mind clear, ears sharp and hands off until the trunk of the fetus has passed through the vulva ring.

If the breech comes into the pelvis and the membranes are unruptured, the further we keep away from the patient and the longer we can keep away from the house, the better it is for the baby and for the mother. We may use morphin

* This lecture was delivered on Oct. 27, 1922.

with fair freedom in such a case. Three points must be remembered in order that a breech may be born spontaneously: 1. *We must have absolute dilatation of the cervix; the cervical ring must be paralyzed;* and we cannot paralyze the cervix except by the continued presence of the bag of waters or the fetal body. The longer the fetal body remains in the cervix, the better it is and the easier is the subsequent delivery of shoulders and head.

2. We must keep our hands off, because every traction makes deflexion.

3. Don't pull. Let the woman push. *You guide;* and here is where the intelligence comes in.

These are the fundamental principles in the treatment of a simple breech: full dilatation of the cervix; paralyzing the cervix by maintaining the bag of waters; keeping the hand out of the woman's vagina until the trunk is born; and, finally, don't pull, but let her push and you guide the body along the lines of the natural mechanism.

In every breech case we are dealing with three distinct mechanisms: First, the mechanism of the breech; second, the mechanism of the shoulders; third, the mechanism of the after-coming head. Each one of these successive parts of the fetus must come into the pelvis, strike the levator ani muscles and be rotated in the pelvis. In the case of the breech this is followed by lateral rotation and external rotation as the shoulders enter the brim, for they must enter in the transverse or oblique diameter. Hence, the back must be rotated so that it may come toward the front, in order that the shoulders can properly come into the brim. Finally, there is the mechanism of the after-coming head, which follows a mechanism similar to that of the fore-coming head, except that the other end of the avoid, the mental process, comes first.

Repetition in teaching is important to impress sequence; therefore, I want to emphasize above all else that every breech case has three separate and distinct mechanisms: that of the breech, the shoulders and the head; that in each there is descent, engagement, rotation and then lateral rotation of the body after the shoulders descend and rotate; the head then enters with the long axis of the ovoid in the

long axis of the pelvis, the chin comes down, strikes the levator and is rotated toward the front.

When the breech reaches the pelvic floor, the anterior hip or the posterior hip will show first at the vulva. In most cases I have noted that it is the anterior hip that shows first, if the woman has a good pelvic floor. Frequently the anterior hip comes down first and is pushed back under the pubis, while the posterior is shoved over the perineum. This brings the bitrochanteric diameter out in the anteroposterior of the outlet. If we leave expulsion to nature, we will notice that the body will rotate and the back will come more and more toward the front. Why? So that the shoulders can adjust themselves in the brim of the pelvis, and as they do this, they go through exactly the same mechanism as did the breech; this again is followed by that of the head, which successively goes through flexion, descent, internal rotation and finally, expulsion.

In the normal case, therefore, understand that the breech is able to take care of itself with the help of your intelligence, *not by your pulling;* that the basic principles are preservation of the membranes, time, and morphin for dilatation.

We now come to the consideration of version and the more difficult cases of breech.

I will quote from Dr. Potter's address before the Philadelphia Obstetrical Society. In this report he states that he delivered 1113 women in the course of one year. This represents a very large amount of work, as we can readily see. In this series he did 920 versions and 80 cesarean sections—an average of one section in every 14 deliveries. There were 12 cases delivered spontaneously before the physician arrived, and 10 vertex cases which were too far advanced in labor to have the head pushed back, of which Potter very aptly says: "I was robbed of 22 operations by these spontaneous deliveries."

In contrast I will record the operative incidence in 1000 consecutive cases where prenatal work has been done. These cases were delivered by my residents, not by an expert. There were 43 operative procedures in 1000 deliveries:

22 low forceps,
5 versions,
8 cesarean sections and
8 correction of malpositions.

Potter said to me one day: "How do you expect to know anything about version if you do only so few?" That is perfectly true, but we haven't found the necessary indications.

You will naturally ask me whether his results justify such frequent operative interference.

In our series the infant mortality, including the babies that died within fourteen days after birth, was 25, or 2.5 per cent. There were 19 stillbirths, and 6 babies died as the result of prematurity or syphilis within fourteen days; or 1 in 40. Potter had 67 infant deaths in 1000, or, to be accurate, 74 in his 1100 cases, or 1 in 14. In other words, proper prenatal care combined with let-alone, commonsense obstetrics will give us practically 3 healthy babies against 1 if we follow Potter's indications for interference.

We now come to the consideration of breech cases that are not normal, with the membranes ruptured and the breech still at the brim.

Most of these are what are known as frank breeches; that is, the legs are extended and closely applied to the body, instead of being flexed on the thighs. This complication makes the trunk more rigid and interferes with its adaptation to the pelvis.

It is a fundamental principle in the management of the breech where the child and pelvis are relatively normal, that if the breech has not come into the brim, the cervix is undilated and the membranes are ruptured, that the breech be broken up and a foot be brought down so that we may have a handle to deliver with in case of emergency. The broken breech is more easily delivered than the frank breech, for in a frank breech the legs are so closely applied to the body that the trunk is rigid and the normal mechanism is disturbed.

When it is necessary to break up the breech, the first maneuver in bringing down a foot is to introduce the hand into the vagina and pass two fingers through the cervix into

the uterus and up between the thighs. The anterior thigh is then pushed out from the body, which allows the leg to flex on the thigh and brings the foot within reach of the two intrauterine fingers, for as the knee is pushed outward, the foot drops within reach of the fingers, and a finger can be slipped over the foot, and the foot drawn down through the os. This makes a wedge-shaped dilator of the breech and diminishes its bulk, and owing to the fact that the membranes have been ruptured and the water drained away, the uterus closely adapts itself to its content. Therefore, this maneuver can only be done under full surgical anesthesia.

The second class of complicated breech cases is that where the frank breech, because the baby is a little smaller, is driven down into the pelvis, but, owing to the rigidity of the trunk, its descent and rotation are arrested, either in the cavity or on the pelvic floor, and we have what is called an impacted breech. These cases usually come so far down in the pelvis that they cannot be dislodged upward and broken up, even under complete surgical anesthesia, for the uterus is firmly moulded about the fetus and grasps it with a retraction ring; hence the delivery must be accomplished by traction from below—but how to make this traction is the question. The forceps is not designed for or adapted to the breech, no matter how applied. Therefore, if the breech is in the pelvis, it can only be drawn down by a fillet, or by hooking the fingers in the groin. Formerly the fillet was a device of common use, but of late years it is seldom seen. The double fillet is the most efficient tractor and is the easiest one to apply. It can be made from an ordinary roller bandage doubled and twisted into a rope with a knot tied in each end. After being sterilized, one end is well lubricated and passed over the groin from the outer side of the thigh and brought down between the thighs. The other end is passed in a similar manner from the outer side of the other thigh. When both ends are out of the vagina, the loop is slipped up over the sacrum of the child. This assures a safe purchase for traction and we are less liable to break a femur than when the fillet is passed over one groin. Of course, the traction is made in the pelvic axis.

In considering version, two methods deserve description: 1. Bipolar or the Braxton-Hicks version (the two-handed method of doing version), which has its largest field in the treatment of placenta praevia, the advantage being that the cervix does not have to be fully dilated and the membranes do not need to be ruptured, or they may have just ruptured. The further advantages of this method are that there is less danger of separating the placenta and less danger of sepsis than when internal version is performed.

The first step in a bipolar version is to introduce the hand into the vagina and two fingers through the cervix, while the other is placed over the breech. The vaginal hand displaces the presenting part, tossing the head in the direction towards which the occiput points. Therefore, it is necessary to know the position of the head, the position of the back and the location of the feet, for a baby can best be turned in flexion through the smallest circumference. We can therefore turn through a smaller arc if we carry the breech in the direction of the feet, and the head in the direction of the occiput. In the interval between pains the head is pushed up into the iliac fossa toward which the occiput points, and held there during the pain. In the interval between this pain and the next, the external hand will carry the breech in the direction of the feet, while the head is held out of the pelvis with the vaginal fingers. Not until the presenting part is displaced into the iliac fossa and held there, can we attempt to turn the rest of the child. By a succession of the foregoing steps, the feet are brought around within reach of the fingers passed through the cervix, when the fingers may be passed over the feet, drawing them through the cervix. A version is never completed until the knee or both knees are outside of the vulva, because until the knee is out, the breech has not entered the brim of the pelvis and been engaged.

An absolute contraindication to version is *a tightly-moulded uterus, with the formation of a retraction ring*, with the greater part of the head or the entire head passed through the cervical ring. This is an absolute contraindication to version, no matter whether we are Buffalo, or Brooklyn, or New York. Why? Because we cannot displace the head back into the uterus through this retraction ring without

rupturing something, and it is usually the uterus or the uterovaginal attachment. The next contraindication is a pelvis which is too small to allow the passage of the after-coming head.

The indications for version are numerous. Potter found an indication for 920 in 1113 deliveries. Apparently he considered that every case where the cervix was sufficiently dilated for him to pass his hand through, irrespective of the position of the head, was an indication to turn the baby. By doing so he saves the woman the pain of the second stage. The woman is unconscious. She believes that she has had a painless delivery, but she does not realize that the chances are 1 in 14 that she will lose her baby, or that one woman out of every 14 will have a cesarean section.

In doing an internal version there are certain conditions which facilitate its execution:

1. The position of the patient. The patient should be on a table, the hips drawn well to the edge, thighs semi-extended and abducted, with the legs partly flexed on the thigh. The limbs may be held in this position by two assistants.

2. The pelvis must not be *so* contracted that the after-coming head cannot come through it.

3. The uterus must not be spastic.

4. The membranes should be unruptured, if possible. However, one can do an internal version if the waters are absent just as well as if they are present, provided we have relaxation of the uterus by complete surgical anesthesia.

Odd as it may seem, there is but one drug that will give complete uterine relaxation, and that is chloroform—not ether and not gas and oxygen. Chloroform, properly administered, will give us absolute relaxation of the uterus as long as no retraction ring has formed.

Formerly it was a matter of great moment which hand should be used in performing version—whether it was done with greater facility with the right or with the left; and rules were laid down to guide us in the selection of the hand to be used. Experience has taught us that neither has any great advantage, and that it is well to practice the use of one hand, preferably the left, in all versions, in all positions. Occasionally there is an advantage in posterior transverse cases in

the use of the hand which corresponds to the side on which the breech lies. As a matter of fact, we have learned that the hand is not a very great factor, and that we can turn with the left as well as with the right hand if we familiarize ourselves with the procedure. Potter uses the left hand exclusively.

When we have decided to do an internal version, the first thing that must be done is the aseptic preparation and dilatation of the vulvovaginal orifice. This means its slow dilatation. Beginning with two fingers and plenty of green soap, the vagina and pelvic floor are thoroughly ironed out while the woman is going under the anesthetic, until the hand can be passed through the vulva ring without causing an abrasion in the hymen. This will take from ten to fifteen minutes. This preparation is most important in every case of version because if the vulvar outlet is not properly prepared, we will tear it. This preparation takes time, chloroform, soap and the sterile gloved hand.

After preparation of the vaginal orifice, we pass the hand up through the cervix, which must be sufficiently dilated to permit its easy passage, and on into the uterus, if possible without rupturing the membranes. We try to separate the membranes from the side of the uterus to a point near the fundus, and locate the position of the placenta. As a rule, the fetal surface of the placenta is opposite the belly of the child; hence if the fetus is in an L. O. A. position, the placenta will be on the right wall of the uterus.

The membranes should be ruptured high up. This permits the hand to plug the opening and thus retain the waters, which facilitates the version. The hand is now boldly pushed upward until the breech is reached, and then slides down the thighs, unflexing the legs, and grasps the feet; for I tell you the best thing to do is to get both feet.

In the interval between contractions, if there are any (if the anesthesia is complete, there will be none), traction is made on the feet while the external hand displaces the head upward in the direction toward which the occiput points. Immediately this is done, the anesthetic should be withdrawn.

In my experience and from watching Potter work, I have found that the anesthesia must be so complete that the

uterus is completely relaxed, making it possible to displace the head upward through the uterine wall by external pressure while traction is being made on the feet.

Once in a while we get a neglected shoulder presentation with a prolapsed arm, so impacted in the brim that it is difficult to displace it. In such a case it is wise to place a noose on the arm before attempting the version, so that when we come to deliver the after-coming arm, we can, by traction on the tape, bring the arm down alongside the body. The objection to this procedure, however, lies in the fact that as the noose is drawn up during the version it may pass over an unprotected rectum, increasing the possibility of infection.

To come back to delivery. Stop the chloroform the moment the version is completed and also stop all traction as soon as the knee is at the vulva; for then, and not until then, is the breech engaged. Wrap the legs in a warm cloth or put them back inside the vulva and sit down quietly and let the patient alone. Why? Because the cervix must be paralyzed and the child must adjust itself to the diameters of the pelvis before extraction can be begun. There is no hurry at this time and in the course of a few minutes active expulsive labor pains will occur, and the delivery will be accomplished without traction. All it needs is your intelligent guidance.

As in ordinary breech deliveries, the child is in no danger until the umbilicus is out, for not until then is there any interference with the fetoplacental circulation—it is only when the body has escaped from the cervix that the cord is grasped by the cervical ring and compressed between the thorax and the cervical rim. The mechanism of delivery is exactly the same as has already been described in our consideration of breech. Don't pull, let the woman push; you guide, keeping the fetal dorsum towards the front. Allow the body to lie in the paralyzed cervix; and by keeping the hand against the abdomen of the child we can keep track of the pulsation of the heart and so keep "tabs" on the fetal heart. The thing to remember is that these breech cases, if the cervix is sufficiently paralyzed, will come through with your guidance, not by pulling on them. The fetal dorsum must be kept toward the front, and not until the navel

has passed the vulva is there ever any need for traction. Potter claims that suprapubic pressure interferes with the maintenance of flexion. He says that normally the head is flexed on the chest, while the arms are flexed across the chest of the fetus and are held in this position by the flexion of the head. This flexion is maintained as the uterus follows down during expulsion, for it is closely applied to the fetus during contraction. Hence there is no need of suprapubic pressure. As the shoulders pass through the brim, the body rotates into the anteroposterior diameter of the outlet and is expelled through this diameter; hence the weight of the body as it drops backward over the perineum exposes the anterior shoulder, which may readily be dislodged by passing the index finger over it and hooking it down under the pubis. When the anterior shoulder is delivered we use it as a rotator, grasping it between the thumb and index finger, to bring the posterior shoulder around to the front, which allows it to be easily dislodged in the same way. If the above mechanism is followed the uterus will have contracted down upon the after-coming head so that it will have its flexion maintained and follow the shoulders into the pelvis—without any help from you.

Delivery of the after-coming head is accomplished in the following manner: The uterine contractions have driven the head with its mental process leading into the pelvis with the child's trunk lying on the operator's forearm; the palmar surface of two fingers of the same hand are passed into the vagina and placed against the canine fossae or into the child's mouth. This increases the flexion. Two fingers of the other hand are then hooked over the shoulders astride the neck for traction which is made downward and backward, then upward and forward until the child's mouth has passed over the perineum, which means that the frontomental diameter is in the vulva ring. After the face is outside of the vulva, the fetus can be held up by the feet while the rest of the head is spontaneously delivered, an ideal position for resuscitation. All we need to do is to hold the child up by the legs and milk the trachea and the mucus will run out of the mouth. The face will then become blue and the fetus

make a gasp and finally breathe, due to a stimulation of the respiratory centers in the medulla oblongata.

Don't worry over blue babies. The pallid ones are the ones we should worry about. Blue babies, if we will hold them up by the feet, milk the trachea and leave them alone, will breathe spontaneously in spite of us. After proper preparation of the vaginal orifice with sterile soap, your hand, chloroform and time, it is not necessary to cut through these pelvic floors in breech deliveries; and it is surprising how few of them are injured if we will but take time after the face is delivered to let the head come out, by means of the expulsive efforts of the patient.

ECTOPIC GESTATION*

JOHN OSBORN POLAK, M.D.

THE question which Dr. Welton has asked me to consider is the subject of ectopic. I will attempt to show how to make a diagnosis of ectopic in the non-tragic stage, and how each symptom is produced by the pathology which is taking place within the tube. It is difficult sometimes to memorize symptomatology, but never to understand it if we can visualize the pathological picture.

The interesting point to me about ectopic is that twenty-five or thirty years ago it was common to see septic hematoceles, but their cause, the primary tubal pregnancy, was not diagnosticated. Any man should be able to make a presumptive guess, and a pretty reliable one, in ectopic, and it is for this reason that I believe each one of us ought to brush up on the cardinal symptoms.

Our work represents the study of a long series of cases, and we have learned that there are certain definite facts which come into the history. There are certain types of women who are prone to have ectopic pregnancy, while other types are but occasionally affected. There are three different classes of these women. The *first of these* is the woman who has had a previous infection. This infection may have been of gonorrheal origin, it may have been a post-partum infection, or it may have been an infection following a suppurative appendicitis with peritonitis which has given rise to peritubal adhesions—something which interferes with the transit of the ovum. You know that in order for fecundation to take place, there must be an ovum, a spermatozoan and an avenue of transit for the spermatozoan. Unfortunately the lumen of the avenue of transit does not need to be as large for the spermatozoan as for the passage of the ovum. Consequently, often after inflammatory conditions, the first evidence that the tube has regenerated is an

*This lecture was delivered on April 20, 1923.

ectopic, for unless the abdominal ostium is closed the tube regenerates to a certain extent and a lumen of sufficient size is formed to permit the passage upward of the spermatozoan, but not large enough to permit the transit downward of the fecundated ovum.

Hence, the first class of women in whom we find an ectopic are those with a previous history of definite pelvic inflammation following marriage. This may result from a gonococcic infection or from intrauterine instrumentation, as for example, a dilatation and curettage which was not indicated; or, abortion or childbirth; or intraabdominal operations, such as an operation for appendicitis followed by suppurative peritonitis. A large proportion of cases fall under this head.

The *second class* of women are those with a definite history of dysmenorrhea. These are usually women of the hypothyroid type with congenitally deformed structures. Their tubes are longer than the ordinary woman's tubes. Their uterus is smaller than the ordinary woman's uterus. Their cervices are small and conical, their pelves are not infrequently of the funnel type, while the vault of the vagina is narrow. This type of woman will seek the advice of a physician for sterility; he will do something for the sterility and she may become pregnant, but because of the length of her tubes, or their increased tortuosity, the ovum, when impregnated, becomes arrested, and we have the second class of cases which are the next most frequent in point of occurrence.

The *third* and final group of women are those who are always becoming pregnant. For instance, in Barbados a few days ago, I went into a hospital where the surgeon told me that he was operating upon 2 or 3 cases of ectopic a week. They are the type in which the ovum grows too large to go through the tube.

The next point to which I wish to call your attention is the location of the arrest of the ovum and just what happens in each case when the arrest occurs. Figure 109 is a rough diagram showing the three principal points of arrest. These points of arrest are commonly at the neck of the tube, the ampulla, or at the isthmic or in the interstitial portion. The interesting point about this is the frequency of this relation. Arrest in

the uterine end of the tube is the least frequent, only about 3 per cent; arrest in the middle or isthmic portion of the tube is the next most frequent, 21 per cent; whereas 76 per cent of the cases are arrested in transit through the tube at the outer end, and are ampulla pregnancies. This is something to be remembered, because interstitial pregnancy is very dangerous, while isthmic pregnancy is less so; and ampulla pregnancy is least dangerous. The latter produces a symptomatology which can always be made out if one will learn the clinical picture in the non-tragic stage. Seventy-six per cent, the great majority of these cases, fall in the least dangerous

FIG. 109. The three most frequent locations for the arrest of the fecundated ovum in its transit through the tube.

group and are the ones in which you can make the diagnosis without question.

Another point to which I wish to call attention is that ectopic may occur in the stump of a previously amputated tube. The first case of this type that came under our observation was in the service of my former associate, Dr. Sewell Matthewson, about twenty years ago. Another occurred in the service of Dr. Edgar, of New York City. The excellent description of these two cases is on record in medical literature. One can readily understand how such an anomalous implantation may occur by reason of the fact that in throwing the ligature around a tube and tying it off at its base, the mucosa may extrude itself beyond the uterine ligature and prevent the closure of the tube. Excision of the

end of the diseased tube, in doing salpingectomy, prevents the possibility of this occurrence.

Another important etiological factor is the effect of some of the retroversion operations on the lumen of the tubes. This is especially true in this city, where they seem to be the fashion, and no judgment is used as to which retroversion operation is selected. Not infrequently in these cases the tube has a short meso salpinx; and where such is the case, and the round ligament is brought up through the abdominal wall, one not infrequently angulates the tube. In this study there were 8 cases in which a previous Gilliam or Baldy-Webster operation had been done, which had so angulated the tube that arrest of the fecundated ovum had taken place. Therefore, beware of cases of anomalous hemorrhages that give a history of a previous retroversion operation. There may be an ectopic in that tube.

CARDINAL SYMPTOMS OF ECTOPIC

1. A history of a missed, delayed, prolonged or anomalous menstruation.

2. Following this a characteristic, bloody vaginal discharge—reddish brown— mixed with mucus that does not clot.

3. Sudden, sharp, colicky, abdominal pain referred to the region of the fruit sac or to the epigastrium, which may or may not be attended by faintness.

4. Extreme sensitiveness in moving the cervix.

5. An enlarged, empty uterus, slightly displaced upward, forward, or to one side.

6. A sensitive, fluctuant, growing mass in the cul-de-sac with increased pulsation of the uterine artery on that side.

8. A slight elevation of temperature—99° to 100⅘°—without other evidences of pelvic inflammation.

9. Repeated attacks of abdominal pain when under observation, at rest, without explainable cause with coincident increase in size of tumor.

I will now attempt to explain each symptom on its pathology. I suggest memorizing the foregoing signs so that in the future, when confronted with a case presenting

these symptoms of early tubal pregnancy, you will be able to make the diagnosis before the tragic stage, and thus it will be possible to operate without risk to the patient.

If the woman has a missed menstruation followed by anomalous bleeding, with or without a sudden attack of abdominal pain, we naturally think of a threatened tubal abortion. It is important to remember that the majority of tubal pregnancies terminate in tubal abortion, and that this abortion may be complete or incomplete, just as when the pregnancy has taken place in the uterus, persistence of the anomalous bloody discharge always suggests the possibility of tubal abortion, for as in intrauterine abortion, the bleeding continues until the pregnancy is terminated. Of course intrauterine pregnancy must be excluded by the physical signs of a uterine gestation. Another cardinal sign is the presence of a sensitive cervix, sensitive to the slightest motion. A missed or delayed menstruation, a bloody vaginal discharge and extreme sensitiveness of the cervix when moved are almost always present. In the analysis of these 307 cases, 299 had a sensitive cervix: a cervix in which a touch by the examining finger elicited a wince, even when the patient's attention was distracted. We next come to the consideration of the tender mass on one or the other side of the uterus. This mass is always sensitive and rapidly growing, because of the accumulation of blood within the tube; the blood escapes from the free end, gravitates into the cul-de-sac and excites a certain reaction in the peritoneum, and this reaction in the peritoneum causes a slight temperature. All tubal abortions have a rectal temperature of 99½° to 100.4°F. This slight temperature in the non-tragic stage is the result of a biochemical reaction of the blood on the peritoneum. The blood necessarily gravitates into the Douglas sac and causes a reaction in the peritoneum covering the uterosacral ligaments; these become tender and spastic, and touching the cervix gives pain.

Naturally we cannot have any reaction without some change in the tissue—a cellular reaction; consequently, there must be a leucocytosis. Hence the sensitive cervix, the slight elevation of temperature and the leucocytosis are due to the effect of escaping blood on the peritoneum. Finally,

when the woman is at rest in bed and not receiving any treatment except perhaps codeine and a low enema, *she will, if she has an ectopic, have exacerbations of abdominal pain, and increase of her bloody discharge, slight elevation of temperature and evidences of a sensitive cervix, while the mass on the side of the uterus shows progressive growth.*

These are the classical symptoms, the cardinal symptoms of an unruptured ectopic pregnancy. In making a diagnosis the most important point of all *is the history*—not what we feel—and then the interpretation of what we feel, combined with the history. Women are peculiar. They have certain rights. They may change the time of their menstrual periods and yet be perfectly normal. One woman may menstruate every twenty-eight days, and then after having a baby, or some operation, have a perfectly normal menstruation every twenty-one days. Or again, a woman may be perfectly normal and flow every thirty or thirty-one days. In the hypothyroid type of women, not infrequently there is a longer interval—six or seven weeks—but there is always some definite periodicity which brings the woman within the realm of normalcy. Abortion is the most common cause of uterine bleeding during sexual maturity. Abortion occurs, as a rule, at the time when the woman would naturally expect her menstrual period, most frequently at the second or third month, just about the time that the placenta forms. The bleeding in abortion is characteristic. The hemorrhage is more profuse than the vaginal bleeding of ectopic. It clots and is continuous until the uterus is completely evacuated. On the other hand, in ectopic, unless the pregnancy is interstitial, the tube always ruptures or the pregnancy aborts before the eighth week, with a coincident menstrual anomaly, i.e., vaginal bleeding.

Tubal abortion, the most common form of ectopic pregnancy, always gives a typical menstrual history, yet there may be no pain. The woman who has always menstruated regularly either anticipates her period by a day or two, or the period is delayed a few days, when she begins to flow and the flow continues usually without pain, *and the discharge is characteristic (reddish brown in color) mixed with mucus, and does not clot.*

All the symptoms in tubal pregnancy are a result of the ovum implanting itself on a surface not protected by a normal decidual reaction. Nature provides a true decidua in the uterus, that may be compared to a thick Wilton carpet protecting a polished hard-wood floor. In other words, the decidua is a protective covering for the uterine muscle. The ovum erodes itself into this decidua, but the underlying structures are not invaded by the erosive action of the trophoblastic cells covering the villi because of the thickness of this protective layer. These syncytial cells with their

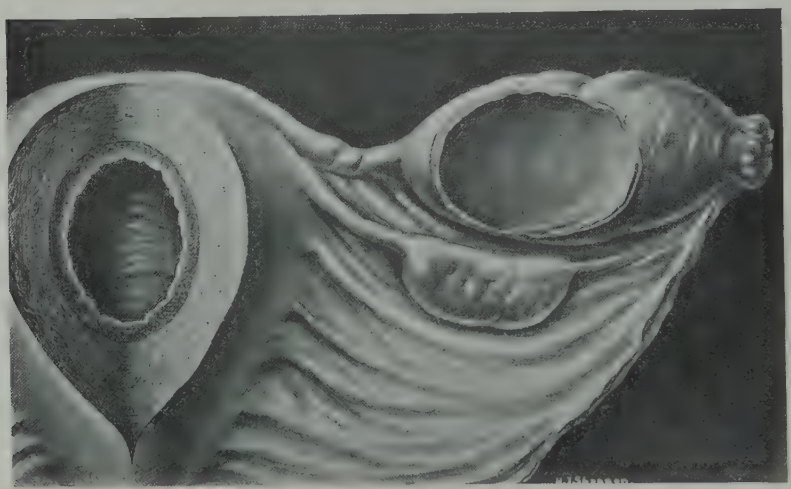


FIG. 110. Drawing from specimen in author's collection showing perfect decidua in uterus in intrauterine pregnancy, and *imperfect decidual reaction* in tube in a nine weeks' tubal pregnancy. This imperfect reaction is the basic cause of the symptoms in tubal abortion and ruptured ectopic.

erosive powers have no respect for tissues. They have been found in the lung in pregnancy because some of them have eroded through into a blood vessel and reached the lung through the general circulation. They have also been found in the brain following pregnancy. On the other hand, when the ovum implants itself in the tube, it does so on a surface where there is no complete decidual reaction. There is no real decidua in the tube similar to that in the pregnant uterus, yet we have an ovum with the same villi, with the same erosive power as in the uterus, and every symptom of

PLATE III.

Tubal abortion; drawn from specimen. (Long Island College Hospital.)

ectopic is dependent directly or indirectly on the absence of a true decidual reaction.

When an ovum locates itself in the tube, it erodes itself into the tube wall, gets its nourishment through the villi and grows, distending the tube to several times its normal size. The tube, as you know, is made up of muscular structures of the same kind that we find in the intestine (a muscle that contracts); consequently, as it is distended by the growing ovum, it is constantly attempting to expel its content. This explains the colicky pains in the lower part of the abdomen in tubal pregnancy even before any rupture has taken place. It also explains the other symptom that the woman frequently complains of, namely, the sense of soreness. Why soreness? Because the tube is stretched beyond its capacity. The rapid growth of the intratubal mass is not due solely to the development of the ovum, but to bleeding into the decidua produced by erosion of the villi into the tube wall with its numberless venous radicals. The trophoblastic cells penetrate the muscle and imbed themselves in it, striking small blood-vessels and causing hemorrhage. This hemorrhage takes place into the decidua and partially unseats the ovum. As it continues, the ovum becomes completely separated from the decidual bed, and abortion occurs. Sometimes the ovum is entirely surrounded and compressed by blood clots.

When the intratubal pressure becomes too great, due to the accumulation of blood behind the ovum, the blood, or the ovum, or both, may escape through the abdominal ostium. The blood that escapes from the free end of the tube gravitates into the cul-de-sac and excites a reaction in the peritoneum covering the Douglas sac. This blood also gives rise to a reaction in the utrosacral ligaments, with resultant sensitiveness of the cervix. This blood, by its biochemical reaction on the peritoneum, is what gives us the leucocytosis and the slight elevation of temperature. As the tube increases in bulk it increases in weight and drops down beside, in front of or behind the uterus, gradually displacing it. The mass in the cul-de-sac increases in size as more and more blood escapes from the dependent free end and accumulates in the pelvis and increases the displacement of the uterus. This

pelvic mass is exquisitely sensitive, owing to the reaction in the peritoneum.

The next form of ectopic that I wish to speak about is the next most frequent in point of occurrence—pregnancy in the isthmic portion of the tube. The pathology is the same, but owing to the location and the anatomy of the tube at this point, the ovum either dies, with a resulting tubal abortion, or grows until the tube ruptures, usually not later than the eighth week. This rupture may be into the peritoneal cavity, with all the signs of the tragic stage or downward, between the folds of the broad ligament, where it may die and become a hematoma or continue to grow and develop as an intraligamentous pregnancy. A point I wish to bring out is that the primary rupture of the tube is preceded by sufficient symptomatology to enable a capable clinician to make a diagnosis in 85–90 per cent of the cases before rupture actually takes place. The intratubal pathology produces the train of cardinal symptoms with which you should all be familiar, and always a sufficient number of them are present before rupture to make possible a presumptive diagnosis. The tube ruptures by an erosive process, and this is slow, preceded by danger signals. When rupture takes place, we have the symptoms of the *tragic stage*, an abdominal calamity. These are sudden pain and usually hemorrhage from the vagina. There is always some degree of shock, with increased pulse and lowered blood pressure. It is then that we get our drop in temperature, and not in the so-called chronic cases. The majority of cases are now diagnosticated before rupture takes place. When that occurs, the question of whether the patient will die or recover depends on the location of the rupture and its proximity to the larger blood vessels. Experience has taught us that if the patient is going to die from primary rupture she will die before surgical aid can be given her.

When an ovum is expelled from the free end of the tube it may die in the peritoneal cavity and become a mole, only to be discovered years afterward when the abdomen happens to be opened for some other condition; or it may retain its placental attachment in the tube and develop as an abdominal pregnancy, oftentimes going to term. We feel that all

abdominal pregnancies are primarily tubal. The diagnosis of abdominal pregnancy is not difficult. There is always a history of pregnancy, a certain number of weeks of amenorrhea, sudden pain and vaginal bleeding; perhaps some attendant shock. Following this the abdomen grows progressively larger and presents certain characteristics: the veins on the abdomen become excessively enlarged; the patient's color is always pasty. On palpation the fetal parts are more easily detected. There is no contracting wall between the fetal parts and the abdominal wall. The fetal heart and the fetal movements are more distinct, and on bimanual examination two separate tumors may be demonstrated. The uterus is always enlarged, but not proportionately so. The cervix is open and admits the finger, and of course the uterus is empty, and displaced usually upward and forward by the fetal mass.

Finally, there are always two tumors in these cases—the tumor of pregnancy and the tumor of the uterus. The uterus is always empty, but it is always enlarged. These make up the diagnostic signs of abdominal pregnancy.

There are thousands of cases of ectopic that we do not recognize and that nobody knows about, that are completely absorbed and no trace of them is left—such is the power of the peritoneum. However, we do not trust to its protective powers alone, if we can make the diagnosis of an ectopic pregnancy.

We now come to the consideration of the interstitial type of pregnancy, which is the most serious type that we have to deal with. This is owing to the fact that the interstitial portion of the tube is surrounded by uterine muscle capable of distending and of hypertrophy, hence the pregnancy goes to a more advanced period of development and has a greater blood supply before rupture takes place. Therefore, when it does rupture, and this always occurs before the sixth month, usually no later than the third, it ruptures in a tragic way. It is said that some interstitial pregnancies terminate as intrauterine pregnancies being expelled into the uterus. Others go on and rupture into the broad ligament with more or less hemorrhage. The majority of them, however, rupture through the top of the uterus, and actually blow its top off exactly as the head of a cylinder is blown out;

and because of the large size of the surrounding blood-vessels these ruptures are extremely fatal. The diagnosis before rupture can be made, but the condition is frequently confused with intrauterine pregnancy. There is the usual history of pregnancy, cessation of the menses for two or three months, but there is always uterine pain *and one or the other cornua of the uterus is exquisitely sensitive.* The uterine colic is frequently attended by slight vaginal bleeding; this is intermittent, as is the colic. The cervix is soft and sensitive, the uterus enlarged and asymetrical, one cornu being more developed than the other and exquisitely sensitive. All these signs are lost by examination under an anesthetic. A patient with such a symptom complex should always be placed in the hospital for observation and be reexamined at frequent intervals. Repeated colics with bloody discharge, and a uterus in which one cornu remains intensely sensitive, should always be looked upon with suspicion.

A woman who has once had an ectopic frequently has another, particularly if her pelvic organs are of the congenital type—the woman with the small vagina, the small cervix, the small uterus and the funnel pelvis. Such a woman not infrequently has an ectopic on the other side. So also does the woman with chronic pelvic inflammation, because a chronic gonorrheal infection is always bilateral; but one tube may recover first. She may have an ectopic on each side at different times, or once in a while at the same time. If she once has an ectopic, she is always subject to ectopic. Having one tubal pregnancy does not immunize her from another ectopic.

Now, we come to the question of treatment.

There is no question that with a knowledge of the classical symptoms produced by the actual pathological changes taking place in the tube, it is possible in the vast majority of instances to make a diagnosis; furthermore, we have the comfort of knowing that the majority of these cases are tubal abortions, complete or incomplete, and thus we have the opportunity of making the diagnosis in the non-tragic stage. I beg of you that every woman with anomalous bleeding, who has a sensitive cervix, where you are not quite sure whether it is a re-lighting of an old pelvic inflammation or ectopic, be

taken into a hospital, placed at rest and kept under observation for a few days. If it is an ectopic, it will grow, she will continue to bleed, and will have other exacerbations of pain. If you are not sure, make a hole in the cul-de-sac and see the blood; or do as my friend, Prof. Reuben Peterson, has suggested—aspirate the mass with a large aspirating needle through the cul-de-sac. I am frank to confess I do not have much faith in this procedure, neither have I the courage to use it. I would much rather make a hole in the cul-de-sac and see. When a diagnosis is made, remove the tube, and, if possible, save the ovary.

In the non-tragic stage the only thing to do is to remove the tube. Whether we remove the tube or incise it and remove the pregnancy, depends on one thing; and that is the condition of the other tube. A large number of tubes are sacrificed by taking them out, merely because our forefathers did so. Although the procedure of splitting the tube, which has been practiced on the other side of the Atlantic for a long time, has been decried, I believe there is some value in it. Why shouldn't we open the tube and empty out the pregnancy, just as you and I would empty the uterus of an abortion, and allow the tube to remain if the tube is normal or approximately so? You may ask, how can you tell it? I must frankly say I cannot tell, but I hope to be able to do so sometime. The next point is, if you take off the tube, there is no reason for sacrificing the ovary (I am speaking now of the non-tragic stage), provided you maintain the circulation of the ovary, not by tying off the tube *en masse*, but by tying off the individual vessels in the mesosalpinx of the tube.

With respect to treatment in the *tragic stage*, if the patient is going to die from the primary hemorrhage she will die before surgical aid can reach her. This was shown in a case where the rupture took place in our ward, and the patient was brought to the operating room and died before she could be anesthetized. Another case that I saw with Dr. Judd some years ago did the same thing. I have seen 3 cases die before anything could be done for them. All these women died before surgery could be brought to them, and they were all in places where surgery could have been done. I have, there-

fore, come to the conclusion that if they are going to die from bleeding, it will occur before you get there. Now, if they are not going to die, the question is as to what we should do. The first thing to do is to *get the patient out of her shock*, and there is only one drug that will do this—morphine. Don't stimulate her, because her blood pressure is as low as it can possibly be and that is her salvation, for if the pressure is suddenly raised the clot will be blown off and the bleeding renewed. Apply abdominal compression, by putting a sand bag on the abdomen, give her morphin, raise the foot of the bed, apply heat and sit down and watch the pulse and take the blood pressure. We will find that the blood pressure drops, usually to 70 or 80—I have seen it even go down to 60—and if the bleeding continues, it will continue to go down. On the other hand, if it does not continue, a clot will form, the bleeding cease and the pressure begin to rise, and the pulse will begin to slow down. If we can hibernate our patient for an hour or two, we can then decide whether she is going from bad to worse or from worse to better. If she is going from worse to better, don't operate immediately, but wait for twelve, twenty-four or even forty-eight hours, until she has reacted, so that we can do surgery as it should be done, and not emergency surgery. Should her condition continue to go from bad to worse, in spite of morphin, posture and abdominal compression, immediate operation is imperative. Get hold of a donor whose blood will match, quickly open the abdomen, put a clamp on the blood-vessel, control the hemorrhage and coincidently have her transfused with human blood. In this way we will save more cases of ectopic than by operating immediately on every case we see in the tragic stage.

THE CONTAGIOUS DISEASES*

JOSEPH C. REGAN, M.D.

IN the course of this paper I shall, instead of attempting to cover in detail all the acute contagious diseases, first make some general remarks relative to them as a group, then classify them, and finally deal in some detail and completeness with the symptomatology, diagnosis and treatment of one of the non-eruptive group, namely, diphtheria.

The so-called contagious diseases comprise a special group of the acute communicable diseases. The term "acute contagious diseases" is being replaced by the term "acute infectious diseases," which has a much wider definition and includes a much larger group. It is true, however, that for many of the diseases which I am about to mention, especially the eruptive fevers, the "contagion" is a prominent factor, and is spread with such great ease as to justify the use of the older name, "acute contagious diseases."

These infections are predominantly and characteristically diseases of later infancy and early childhood. They are likely to be regarded by many of us as diseases of relatively little importance.

This viewpoint must be changed, however, for we are beginning to appreciate the fact that many of the cardiac, pulmonary and renal insufficiencies of later childhood and adult life may trace their origin to one of these infections. We are also more cognizant of the devastating effect which they may exert on the nutritional condition and general health of the child, preparing the soil for the development of other infections, sometimes fatal. Finally, municipal, state and federal statistics show that from the standpoint of morbidity and mortality the "Acute Contagious Diseases" still constitute a serious menace to the age of childhood.

* This lecture was delivered on Nov. 2, 1923.

In the control of these maladies we are deviating from former stringent regulations concerning terminal disinfection and fumigation, to a more careful and proper isolation of the patient himself and of exposed susceptibles; the adequate disposal of infected secretions and discharges, or articles soiled by such; and the more careful search for the atypical or acyclic cases, in mild or abortive forms, and the detection of convalescent and healthy carriers of the disease.

We no longer accept such diseases as the inevitable accompaniment of childhood, but are devoting more and more energy to the development of active and passive means of immunization.

The acute contagious diseases may be said to comprise diphtheria, pertussis, epidemic parotitis and the acute exanthemata—measles, rubeola, varicella, variola and scarlatina.

These diseases have certain features in common which may be grouped as follows:

1. They are all caused by living agents or microorganisms of some form (bacteria, viruses, or Protozoa) which have resisted discovery, in all except one or two instances, despite years of bacteriological research and investigation carried on all over the world, even since Pasteur's discovery opened the realm of bacteriology to medical science. It is the concensus of opinion to-day that the diseases of this group, for which the causative agent has not been discovered, are probably due to filtrable viruses.

2. They each have a definite period of incubation, which, while subject to considerable individual variation, is of a fairly constant duration for each disease. With some it is short, as in diphtheria and scarlatina, but for most contagious diseases it is characterized by some length.

3. They each have a fairly typical symptom complex which makes their recognition usually easy, despite the lack of biological and bacteriological diagnostic methods for the majority of them. In this sense these infections are somewhat unique.

4. While generally presenting themselves in the ordinary forms, they not infrequently occur in abnormal, acyclic,

unusual forms, making their recognition in some cases difficult and in others almost impossible.

5. Several of these infections, while relatively benign by themselves, attain a pronounced degree of malignancy in certain cases or certain epidemics because of secondary infections, with which they are so often complicated. One may cite the effect of the streptococcus in diphtheria, scarlet, measles, etc. It seems that the acute infection, by its effect upon the defensive processes of the body, alters the resistance of the tissues, especially the mucous membrane, and activates the habitually saprophytic streptococcus or pneumococcus to an unusually pathogenic virulence. This becomes pronounced in epidemic centers of such infections, where these organisms are transmitted with increasing virulence from one case to another.

6. The acute contagious diseases occur with characteristic frequency in the period of early childhood.

7. Natural immunity to most of them is probably not so common as is apparently indicated by the number of adults who give histories of but one or two of these infections. It is probable that many unrecognized cases of these diseases occur. An apparent natural immunity may really be due to an abortive or an acyclic attack.

8. The person ill with an acute contagious disease is considered to be a source of danger to others in the community. Therefore these diseases are reportable to the Department of Health in the city in which they occur.

9. Certain procedures of quarantine are adopted to prevent their spread, the measures in general being the same, with variations according to the disease.

10. The quarantine period varies in the different diseases and is terminated only after a certain period has passed, or after certain bacteriological examinations are found to be negative, or both.

THE ACUTE CONTAGIOUS DISEASES MAY BE DIVIDED INTO GROUPS: 1. According to whether etiological cause is known:

(*a*) Unknown: Measles, Rubella, Variola, Varicella, Epidemic Parotitis.

(*b*) Known: Diphtheria, Pertussis, Scarlet Fever?

2. According to whether there is a specific preventative or curative therapy:

(*a*) Those with preventative therapy: Diphtheria, Variola, Measles, Varicella? Pertussis?

(*b*) Those with specific curative therapy: Diphtheria, Pertussis?

3. According to degree of contagiousness:

(*a*) Those spread with extreme ease: Variola, Varicella, Measles.

(*b*) Those spread with more difficulty: Rubella, Scarlet Fever, Diphtheria, Pertussis, Epidemic Parotitis.

4. According to whether they are eruptive or noneruptive.

(*a*) Eruptive: Measles, Scarlet Fever, Rubella, Variola, Varicella.

(*b*) Noneruptive: Diphtheria, Pertussis, Epidemic Parotitis.

Diphtheria

DEFINITION. Diphtheria is an acute infectious disease characterized locally by the production of a false membrane almost always on a mucous surface and generally by certain constitutional symptoms and visceral syndromes due to the toxins, which are eliminated at the site of the membrane by the Klebs-Loeffler bacillus.

MANIFESTATIONS OF THE DISEASE. 1. The local sign—the membrane which is the most constant but not the invariable sign of the disease.

2. A general constitutional reaction.

3. Certain visceral syndromes or complications including mainly those of the circulatory, respiratory, nervous and renal systems.

THE LOCAL SIGN—THE MEMBRANE. (*a*) *Formation of the Membrane.* Some minute break occurs in the continuity of the epithelium of the mucosa where the diphtheria bacillus obtains lodgment. It then secretes a toxin which produces degeneration of the superficial epithelium thus producing the first layer of the membrane. The toxin then acts upon the blood-vessel wall causing a transudation of fibrin and of cells which push forward the dead epithelium and in turn are pushed forward from below. When cure supervenes the epi-

thelium is formed beneath the false membrane again and the latter becomes detached.

(*b*) *Structure of the Membrane.* From the superficial to the deeper layers the false membrane is composed of

1. A zone of microorganisms.
2. A zone of dead or dying cells (epithelium, leucocytes) with fibrin.
3. A zone of living but diseased cells with fibrin.
4. The mucosa deprived of its epithelium covering

(*c*) *Main Characteristics of the Membrane.* The most prominent characteristics of the false membrane in diphtheria are the following:

1. A marked adherence to the mucosa beneath so that it leaves a bleeding surface if detached.
2. The property of reproducing itself if detached.
3. The property of being able to spread to neighboring surfaces.
4. The fact that it is localized in the vast majority of cases in pharynx, nose or larynx.
5. The fact that it is insoluble in water and
6. That it produces a mould of the mucosa on which it is located.

(*d*) *Other Characteristics.* The color of the membrane varies from the typical pure white to a yellowish brown or green. The odor is very slight in the pure diphtheria but of a peculiar fetid nauseating kind in the septic forms.

The size varies from a minute piece of membrane to one several centimeters in length and even the entire cast of the respiratory tract. The thickness varies from a thin tissue-paper-like membrane, to one which is 5 to 7 mm. in thickness, firm and inelastic.

CLASSIFICATION OF THE DISEASE

(*a*) *According to whether it is membranous or non-membranous:*

1. Nonmembranous forms—catarrhal and follicular.
2. Membranous form—the usual form of the disease.

(*b*) *According to the localization of the membrane:*

Common localizations are pharyngeal, nasal, laryngeal and tracheobronchial.

Rarer localizations are conjunctival, buccal, vulval, aural, cutaneous and wound diphtheria.

(*c*) *According to the microbian cause:*

1. Pure diphtheria—due to the Klebs-Loeffler bacillus.

2. Septic or polymicrobian diphtheria due to the Klebs-Loeffler bacillus associated with other microorganisms, notably the Streptococcus.

(*d*) *According to the severity of the disease:*

Mild, moderate, severe and very severe.

SYMPTOMS OF THE VARIOUS FORMS OF PHARYNGEAL DIPHTHERIA

Catarrhal or erythematous angina due to the diphtheria bacillus is rarely recognized clinically, and then only from cultures. The importance of these cases, however, is that they are important disseminators of the disease.

Follicular diphtheria is likewise seldom recognized clinically alone. It is usually only when a bacteriological examination is made that the true condition is realized.

In the usual, mild or moderate, pharyngeal diphtheria the disease may be revealed only on examination of the throat. In the mild cases the membrane is usually of a white color and may partly cover one or both tonsils, but there is little redness and little if any edema. In the moderate cases it covers both tonsils and extends perhaps to the anterior pillars, and perhaps presents a small area on the uvula. The inflammatory reaction is slightly greater, and a little more edema is noticeable. The general condition is very little altered. The temperature may be elevated between 38° and 39°C. Some dysphagia may be experienced, and a light degree of local adenopathy is demonstrable on palpation of the submaxillary lymph nodes. Such are the majority of cases that come under treatment early. They represent a pure Klebs-Loeffler bacillus infection upon which diphtheria antitoxin will have a prompt and rapid curative effect. They must be placed thus in contrast with the severe septic group, in which the disease has been neglected, in which inadequate antitoxin dosage has been administered, or in which the infection seems from the onset to be malignant.

In the severe or septic forms of pharyngeal diphtheria we have an entirely different picture. The membrane covers the entire mucosa of the throat extending upward to the soft and even to the hard palate, and backward involving the pharyngeal wall, so that the entire throat is covered. With this there is often a pronounced edematous swelling of the tissues, so that the tonsils almost meet in the mid-line. The membrane is yellowish green, or brown with black areas interspersed. The odor of the breath is a horrible fetid stench that, when once experienced, is never forgotten. The nose is often involved, as evidenced by a profuse bubbling seropurulent discharge which constantly streams from the anterior nares which it excoriates in passing. The neck in its upper portion is greatly swollen, obliterating the normal anatomical configuration of the junction of the face and neck, due to the marked adenitis and periadenitis. This swelling is not hard or indurated, nor is the skin overlying it reddened. The temperature may be only moderately elevated or almost normal, the pulse is thready and often irregular, and urinary analysis often shows a massive albuminuria. The evolution of the disease is often fatal in the first week, due either to the extension to the respiratory tract with mechanical obstruction to breathing, or to the effects of the toxemia. However, under the influence of massive doses of antitoxin (especially intravenous) one sees today a certain proportion of these cases in which the throat is gradually cleared of membrane little by little, the glands subside and the edema of the neck disappears. The improvement continues and after a slow convalescence of weeks or months, in which moderate myocardial or paralytic complications have occurred, the patient eventually recovers. But often one sees supervene a group of symptoms which Marfan has very properly termed the "secondary syndrome of malignant diphtheria" and which are the prelude to a rapid death. The symptoms in these cases seem to be largely of cardiac origin. The onset of this symptom complex is announced by short periods of lipothymia lasting a few minutes, during which the patient is extremely pale and speaks very few words. Shortly after this, bradycardia, with a fall of the pulse to 40 or 50, develops. A cardiac

arrhythmia with partial or complete heart-block may then appear. The liver is augmented in volume, reaching four to five fingers' breath below the costal margin. Nausea and vomiting prevent the taking or retaining of alimentation by mouth. Urinary secretion is diminished. The patient complains of a feeling of profound weakness and even of anguish, and the facies is pale, and bears the stamp of the inevitable fatal termination fast approaching. The extremities become cold, the pulse is almost imperceptible and the heart sounds are more and more feeble until finally, within one to three days, the patient expires in a sudden syncope attack.

VARIETIES OF THE SEPTIC FORM OF PHARYNGEAL DIPHTHERIA

There are really three types of cases grouped under this heading: (1) The usual or typical one has been described. (2) Hemorrhagic form, the true malignant diphtheria. (3) Phlegmonous or peritonsillar form.

2. The hemorrhagic form resembles in all respects the septic type of the disease, but has an additional symptom—the occurrence of cutaneous and visceral hemorrhages. The cutaneous hemorrhages are usually manifested as a scant eruption of minute petechial spots of a reddish, purplish or blue color, scattered here and there over the body, sometimes missed in a superficial inspection. These petechiae have an extremely bad prognostic significance, and cases presenting such an eruption rarely recover. The petechiae are seldom found on the conjunctival or buccal mucous membranes, differing thus from those seen at times in acute lymphatic leucemia, with which the disease may sometimes be confused, unless a blood count is taken, owing to the presence of a necrotic exudate on the tonsils in the latter condition. Occasionally areas of ecchymosis are also present, and usually in these cases hypodermic injections cause a hemorrhage at the site of introduction of the needle. Visceral and mucous membrane hemorrhages are also not infrequently seen in this form of the disease, epistasis and gastrointestinal hemorrhage being the most frequent.

3. The phlegmonous type is of considerable importance, not because of its frequency, but from the standpoint of diagnosis. The disease, when it assumes this form, is frequently considered and treated as a peritonsillar abscess until the process is so far advanced that irreparable damage has been done to the heart. It is not strange that this mistake should occur, for the throat presents very similar pictures in the two conditions. There is the same marked inflammatory swelling and pronounced edema of the peritonsillar tissues with some displacement of one or both tonsils toward the median line, commonly with edema of the uvula and supratonsillar portion of the soft palate, and with very little visible membrane. This often covers at first only one or both tonsils and, owing to the swelling, is partly hidden. It is only as the disease advances and the membrane spreads over the anterior pillars and up on the soft palate that the true condition may be suspected. Perhaps the apparent abscess may be incised, and instead of pus being obtained, the membrane spreads along the site of incision and over the neighboring mucosa.

These cases possess certain characteristic points which differentiate them from a peritonsillar abscess. The swelling develops too rapidly for an abscess, the glands on the affected side or sides are much more swollen than in an abscess, the breath has the peculiar fetid odor of septic diphtheria, there is far less pain and dysphagia, and frequently the presence of diphtheritic rhinitis is indicated by a serous nasal discharge.

Nasal Diphtheria

Diphtheritic rhinitis may be primary or secondary.

Primary nasal diphtheria is a peculiar disease, differing much from typical pharyngeal diphtheria. It often remains latent, showing itself after a short or long period by laryngeal involvement or by paralysis. In such cases the child often presents little or no temperature, and the only sign of the disease is a dark brown semi-liquid discharge which occurs now and then from the anterior nares, with a tendency to excoriation of the nostril, occasional epistaxis and slightly palpable submaxillary lymph nodes. If the condition lasts a

long time, a progressive secondary anemia may develop. The condition is often overlooked unless a nasal culture is taken. Such cases may be a potential source of dissemination of the disease, especially in institutions.

Secondary diphtheritic rhinitis may be benign or malignant. The benign form is usually a pure Klebs-Loeffler infection accompanying a mild or moderate angina, and characterized by slight symptoms and a moderate discharge from the nose.

The malignant form is due to a mixed infection with the Klebs-Loeffler bacillus and Streptococcus, pneumococcus, etc., and accompanies certain of the septic pharyngeal forms of diphtheria. It is characterized by the severe toxic symptoms found in such cases, with a profuse seropurulent nasal discharge which literally "streams" from the nostrils, and contains pieces of membrane.

Pharyngeal Diphtheria

DIAGNOSIS. A bacteriological examination alone secures the recognition of the unusual forms of the disease such as the erythematous and truly follicular or cryptic types. Regarding the latter, certain fine differential clinical points will be mentioned later.

In its usual membranous form the disease can be, and usually should be, readily recognized clinically. Within twenty-four hours of the onset it should be possible, if the throat is examined, to make the diagnosis. It is an important fact for physicians to remember that an inspection of the throat is *always* part of the general physical examination of a child. This is a wise rule to adopt, for many times indeed it has happened that a child with a diphtheritic membranous angina has never complained of a sore throat. I have seen the disease advance to such an extent that the entire throat was a mass of exudate without the child having once complained of pain, and the parents were entirely without suspicion of the condition that existed. Temperature is also a very unreliable guide. Indeed it is a striking thing that in some of the worst cases of diphtheria, constitutional symptoms may be so slight that the patient is up and about performing daily play or work for a period of several days from the onset, often even until within a short time of death. Many of us place

entirely too much confidence in diagnosis of diphtheria on the result of the examination of a culture. This viewpoint is entirely incorrect, for the laboratory examination affords only corroborative evidence. A positive culture confirms a suspected diagnosis; a negative culture must have no bearing if the case is clinically positive. In other words, a diagnosis of diphtheria must be made on clinical evidence alone.

One may be inclined to question the stress placed on this point, but in answer it may be said that many children have lost their lives while the physician waited from day to day the return of a positive culture on a case that was clinically positive beyond any question of doubt. While we delay the membrane advances.

The lack of reliance to be placed on culture is due to several reasons. In the first place, many cultures are taken improperly: a superficial swabbing of the surface of the membrane is taken, and often perhaps the membrane is not touched at all. Then the culture is perhaps planted improperly by a too vigorous swabbing of the media. Finally, after the culture is made, it may be carried for some distance, or be delayed many hours before it reaches the incubator, the exposure (especially to daylight and sunlight) in winter, seriously affecting the vitality of the organisms. Too long an incubation may permit the overgrowth of other organisms, numerous in the throat even at the site of the false membrane, and often more hardy and profuse than the Klebs-Loeffler bacillus.

DIFFERENTIAL DIAGNOSIS. Five types of anginas must be clearly differentiated from diphtheritic angina. These may be mentioned:

1. Vesiculous angina—more especially herpetic angina, to a less extent aphthous angina, and the enanthematous angina of varicella and variola.
2. Pseudo-membranous angina, including pneumococcus and streptococcus angina.
3. Ulceromembranous angina, including Vincent's angina, and luetic angina.
4. Phlegmonous angina or peritonsillar abscess.
5. Cryptic angina and pultaceous angina.
6. Parasitic angina.

In the first group, vesiculous angina, the only one which might be confused with diphtheria is herpetic angina. The onset of herpetic angina is more abrupt than that of diphtheria, the constitutional symptoms are a little more pronounced, and there is acute pain and soreness of the throat. The local appearance is different. Instead of a membrane we have groups of small vesicles in clusters here and there over the pillars of the fauces, the pharyngeal wall, the soft palate, and often the buccal mucosa, which soon rupture, leaving ulcerated areas covered by a grayish coating with a hyperemic border.

In the second group both pneumococcus and Streptococcus angina may be confused with diphtheria. Pneumococcus angina presents itself usually as a shallow ulcerated lesion of variable size on one or both tonsils, with a red hyperemic border and a thin delicate whitish-blue covering which is readily wiped away. There is practically no odor, and smears and cultures readily establish the diagnosis.

Streptococcus angina may have a somewhat similar appearance to that of the pneumococcus. It often presents itself, however, in scarlet fever as a well-formed membranous exudate. In the invasion period of the latter disease the differential diagnosis may be difficult, but soon the strawberry tongue, the punctate erythematous rash and the other symptoms make the diagnosis evident. Moreover the pseudo-membrane in scarlet is darker in color, shows a greater tendency to ulceration, especially along the anterior pillars, and has less odor than a septic diphtheritic membrane. Then again it attains a certain maximum of involvement and remains confined there, rarely extending to the soft palate or even beyond the anterior pillars. Of course it is not to be forgotten that diphtheria may and does occur not infrequently at the onset of scarlet fever, and when there is doubt as to the diagnosis, antitoxin should be given.

Vincent's angina is usually easily distinguished by its slow evolution (the patient often having been sick for four to five days when he applies for treatment), by the less pronounced degree of constitutional symptoms and by the local aspect of the throat. The affection is unilateral, as a rule, and appears as a deep ulceration covered by a yellowish-

gray, thick but soft coating which, as a rule, is easily wiped off and leaves a slightly bleeding base. The condition is not painful and has usually a peculiar characteristic odor. There is little, if any, adenopathy, and other lesions of a similar character may also exist in the mouth. Smears quickly determine the diagnosis showing numerous fusiform bacilli and spirochetes.

Luetic angina rarely appears as a chancre of the tonsil or as an ulcerating gumma. More often it is seen in the secondary stage as a mucous patch. It is then an ulcerating lesion covered by a thin pseudo-membrane with hyperemic borders. The pillars are often involved, but the process does not extend over the soft palate. There is less constitutional disturbance than in diphtheria, other plaques are to be looked for elsewhere in the mouth, and the skin should be observed for the syphilitic roseola. Certainly the Wassermann and cultures will decide any question as to diagnosis.

Cryptic angina is certainly not likely to be confused with membranous tonsillar diphtheria, because here the exudate is follicular or cryptic, is of a yellowish or grayish color, can often be squeezed out of the tonsil by pressure, has a bad odor, and is not membranous in structure but can be readily broken up into small particles on a slide.

Pultaceous angina occurs in individuals with hypertrophied tonsils, who are subject to repeated attacks. Several white spots develop on the enlarged reddened tonsils, which occasionally fuse to form one to two small patches. These spots are less attached, however, than a diphtheritic membrane, the tonsils are more painful and the signs of general intoxication are not so pronounced.

Parasitic angina is scarcely to be confused with diphtheria. The characteristics of the stomatitis elsewhere in the mouth are sufficient to make the diagnosis.

Laryngeal Diphtheria

Laryngeal Diphtheria supervenes, according to some statistics, in as high a proportion as 30 to 40 per cent of all cases of diphtheria, and occurs alone from the onset in about 10 per cent. The symptoms in membranous laryngitis are due mainly to mechanical obstruction to respiration and

secondarily to toxemia. This also has a distinct bearing on the type of complications which follow, as is mentioned later on.

The disease may be primary or secondary. Far fewer cases are truly primary than is generally thought. There may be, for example, a diphtheritic rhinitis with slight local signs, or a pharyngeal diphtheria in which a small piece of membrane is located in an inaccessible portion of the pharynx where it is not seen by examination of the throat; or, finally, the angina may be erythematous but nevertheless diphtheritic. It not infrequently occurs that a follicular angina is found in cases of membranous laryngitis. From the standpoint of diagnosis it is important to remember that the absence of membrane from the tonsils has no bearing in making a diagnosis of laryngeal diphtheria.

SYMPTOMS. These may be divided into four stages. The first three stages are present in all cases but are not always of the same duration.

In the first stage the patient has only a slight huskiness of the voice and a rather hard croup cough, usually with a slight temperature. The attention at this time is often directed only to the dysphonia as evidenced in the cry or in the voice.

Then supervenes the second period, in which an inspiratory stridor appears, with retraction of the chest in suprasternal, supraclavicular fossa between the ribs and of the epigastrium. The pulse increases in frequency, and the aeration of the chest is diminished moderately. All these symptoms become progressively accentuated and the child passes into the third stage. This third stage is of importance because it is the period of operative interference. The retraction of the chest is now marked, the aeration of the lungs is greatly diminished, the pulse is rapid, the radial pulsation falling off with inspiration, and the heart sounds are of poor quality. The stridor is usually high-pitched, piping and shrill, the cyanosis of the lips and fingers becomes evident, and a peculiar pallor appears around the mouth. The child, instead of remaining quietly in bed, now struggles from one position to another, using more and more the accessory muscles of respiration in an endeavor to get relief from the

asphyxia, which will soon supervene if operative interference is not at hand. Spasms occur from time to time, any one of which may carry the child off in an attack of asphyxiation. In a certain proportion of cases not treated, the patient passes into the fourth stage in which he falls back exhausted in his crib, lies in a listless state, with superficial rapid respiration, with little if any stridor, slight retraction and marked cyanosis of the lips and the skin of the extremities. The heart is dilated, edema of the lungs develops, and the child dies in a semicomatose state. The clinical picture in this late period of the disease resembles rather closely that of a child dying of bronchopneumonia, and if the patient is seen first at this time the true condition is readily overlooked.

Mechanism of Production of Symptoms of Asphyxia. Mechanical obliteration of the glottic aperture of the larynx by the membrane and by the accompanying edema plays, no doubt, in many cases a most important rôle. But they are not sufficient in all instances to explain the condition. It is necessary to take into consideration the reflex muscular spasm which occurs from time to time, and which has its origin at the site of the inflamed mucous membranes. The absence of control over the superior centers and the intensity of reflex phenomena are characteristic of the nervous reactions of the child; and indeed, the younger the child, the more important they are.

DIAGNOSIS. There are certain practical considerations relative to this subject which may well be spoken of first.

In laryngeal diphtheria there may or may not be signs of nasal or pharyngeal involvement; in fact a considerable proportion of cases show no membrane in the pharynx. Hence one must not conclude that a case of croup is not diphtheritic in origin because no membrane can be seen. If an exudate is present on the tonsils it not infrequently happens that it is of a follicular type. A diagnosis of laryngeal diphtheria should be made clinically without waiting for a culture return, because the delay of twelve to twenty-four hours entailed in the administration of antitoxin may have serious results for the child. A negative nasal or pharyngeal culture has, moreover, no bearing on the diagnosis. All cases of acutely developing laryngeal stenosis febrile in type and

progressive over a period of several hours should be considered and treated as laryngeal diphtheria unless some obvious reason for the stenosis is known. In doubtful cases, a direct laryngoscopic examination is the only sure means of ruling out diphtheria and establishing with certainty the diagnosis.

LARYNGOSCOPIC APPEARANCES. The larynx is usually moderately congested and often there is redness and slight swelling of the ventricular bands, and at times of the vocal cords. There may also be some edematous swelling of the mucosa at the base of the arytenoides. Pieces of membrane are commonly seen scattered here and there over the arytenoids, under the surface of epiglottis, ventricular bands or vocal cords. The membrane is usually of white color and not very thick, nor adherent to the underlying structure. At times it forms a cast, covering the entire glottic aperture, and at others it is almost completely detached and flaps back and forth with each respiration. Cultures taken from the laryngeal membrane are almost always positive for Klebs-Loeffler bacilli.

DIFFERENTIAL DIAGNOSIS. Laryngeal diphtheria is to be differentiated mainly from the acute forms of laryngeal stenosis; far less frequently from the more slowly developing forms.

(*A*) Acute in onset:

1. Acute infectious laryngitis—catarrhal, ulcerative, and ulceromembranous—as in measles, influenza, pertussis, varicella, variola, scarlet, typhoid, Vincent's angina, erysipelas and herpes of the larynx.
2. Acute adenoiditis and coryza in infants.
3. Stridulous laryngitis.
4. Retropharyngeal abscess.
5. Foreign bodies in larynx, trachea and rarely, esophagus.
6. Bulbar paralysis of diphtheria or acute poliomyelitis.
7. Edematous laryngitis—of renal or cardiac origin, of toxic origin, of nervous origin (angioneurotic edema), of traumatic origin, due to food intoxication (urticaria), or inflammation of neighboring structures (perilaryngeal or tracheal abscesses).
8. Spasm of the glottis.

(*B*) Gradual in onset:

9. Papillomata.
10. Hypertrophy of the thymus.
11. Tuberculous tracheobronchial adenitis.
12. Tuberculous and syphilitic disease of the larynx.

Tracheobronchial Diphtheria

This form of diphtheria of the respiratory tract is far less common than laryngeal diphtheria, but it is extremely grave. It may follow a septic pharyngeal diphtheria, when the symptoms of toxemia, bronchopneumonia quickly overwhelm the patient. Frequently the disease follows a pure diphtheritic angina, when the symptoms of mechanical obstruction to respiration are most prominent.

There is a large number of patients who have no detectible pharyngeal or nasal membrane, who nevertheless present extensive membrane formation in the trachea and bronchi; and these cases are prone to be overlooked.

The late Doctor Henry L. Lynah, whom I had the honor to know well, made a classical study of the disease. He was firmly of the opinion that in a great majority of these membranous stenoses the infection extends from below upward. He based this upon the results of his direct laryngeal and bronchoscopic examination of cases recognized prior to the third day of the disease, in which the larynx was free of involvement, but the trachea and bronchi showed membrane formation. Many of these cases are overlooked and recognized only in the last stages when the larynx is finally involved or a cast of membrane coughed up.

The membrane may occur in the form of a short or a long cylinder (a mould in all respects of the lining of the tracheobronchial tree), or it may be parietal and occupy only a portion of the lumen, appearing then as a long ribbon of variable length. The membrane acts as a valve allowing air to enter the lung more readily than it can make its exit, thus giving rise to certain physical signs which are found.

SYMPTOMS. When the disease starts in the pharynx or the larynx and extends downward we have, of course, first the characteristic signs of pharyngeal or laryngeal diphtheria.

In the group of cases in which the disease starts below, the onset is usually gradual, and aside from an occasional cough

and slight stridor, the condition may be thought to be an ordinary cold, especially as the voice is not lost. Thus the condition may progress for four or five days with the patient up and around, and with little or no temperature. Finally after this insidious progress, the disease begins to show its real nature. The temperature rapidly rises to 102–104°F. and pronounced stenotic symptoms appear, accompanied by cyanosis, rapid pulse, labored and asthmatic type of respirations and retraction.

The physical signs in these cases are suggestive. Inspection reveals a barrel-shaped chest on one or both sides, depending on whether one or both bronchi are obstructed. Cyanosis of the skin, especially of the finger tips, is rather constant. Percussion shows a hyperresonant note over the involved emphysematous portions of the lungs. Auscultation reveals the most constant physical sign, however, in diminished or absent respiratory murmur over the affected side. The râles are of subcrepitant variety or are sibilant and sonorous. Over the trachea in the sternal notch with the head well retracted a "flip-flop" sound may often be heard, due to loosened membrane.

Cases of tracheobronchial diphtheria are apt to escape recognition under the diagnosis of bronchopneumonia. The symptoms are, however, distinctly different. Bronchopneumonia does not have the peculiar blocked expiratory cough, the peculiar progressive asthmatic-like dyspnea, the diminished or absent respiratory murmur with a hyperresonant percussion note. The disease is also to be differentiated from catarrhal bronchitis, foreign bodies and thymic asthma.

TREATMENT OF LARYNGEAL DIPHTHERIA

(*A*) In 1st and 2nd stages—palliative and specific
(*B*) In 3rd stage—specific and operative
(*C*) Operative treatment
  1. The symptoms used as guide
  2. Variations in individual cases
  3. Methods to be used—(*a*) direct laryngoscopy with suction, (*b*) intubation, (*c*) tracheotomy
(*D*) Direct laryngoscopy with suction—description of the method

(*E*) Intubation:—(1) instruments, (2) technique, (3) course, (4) accidents during the operation and subsequent before extubation, (5) methods to be avoided, (6) complications, (7) varieties of tubes

(*F*) Tracheotomy

(*G*) Special considerations regarding treatment of tracheobronchial diphtheria—(1) specific, (2) operative, (3) medication

A. In the treatment of the first and second stages of laryngeal diphtheria the palliative measures to be employed comprise the application of a warm flaxseed poultice to the neck, the administration of small doses of morphine and somewhat larger doses of atropine and whiskey and the use of steam inhalation (not in a croup tent, but in an open room).

The specific measures consist in the administration of diphtheria antitoxin (best intravenously and in proper dosage, a matter mentioned later).

B. In the third stage the question of operative interference comes up. Certain symptoms of this stage must be used as a guide to the need of intubation. It may be said that the most important one thing on which to decide this delicate question is the condition of the heart and circulation. While the color and the pulse are good and radial pulsation does not disappear with each inspiration, and the heart sounds at the apex are of good quality, it is usually safe to wait. On the other hand, where the reverse is the case and there are accompanying symptoms of deep retraction, restlessness and diminished aeration of the lungs, the time for operative interference has arrived. One can err by waiting too long as well as by operating too early. Most patients with laryngeal diphtheria who have good hearts and who previously have been healthy may be allowed to enter well into the third stage before operative interference is adopted. On the other hand, there is a group of cases in which the heart is not good, due to an accompanying pharyngeal or nasal diphtheria, or to the fact that they are recovering from or have another acute illness at the same time, or again that they are affected with chronic nutritional disorders or debilitating diseases. In these patients intubation must be

performed at the end of the second or early part of the third stage.

C. Operative Method to be Used. Formerly our reliance on surgical intervention in these cases was based almost entirely on the use of O'Dwyer's intubation tubes. Recently another method has been devised, which seems to hold out hope of saving more of these children than was possible with the O'Dwyer tube. I refer to direct laryngoscopy with suction.

D. This method, so far as I know, was devised by the late Dr. Lynah for the treatment especially of tracheobronchial cases, in which, of course, intubation often fails to relieve the stenosis. It was later employed, and, with some modifications, put into routine use; first at the Willard Parker Hospital by Dr. Grover and Dr. Hardman—who deserve the credit for developing the method—and later at the Kingston Avenue Hospital by Dr. Dickson and the writer. The treatment has the limitation of being highly technical, requiring an operator familiar with direct laryngoscopy. It is as follows: A laryngeal speculum is introduced by the direct method, and after the larynx is located and in view, a suction tube, attached to a small Sorensen electric suction pump, is introduced into the larynx and, with an easy gentle movement, is passed several times up and down the larynx and upper trachea. It is surprising to see how much membrane may be obtained in this way in certain cases, as is shown when the bottle of the suction pump is examined after the operation. Unfortunately, even though the larynx is free of membrane, a certain number of these patients have to be intubated, owing to edema or spasm.

E. Intubation. This operation was devised by Bouchut, and perfected by O'Dwyer. The instruments used are an introducer, an extractor, tubes of various size according to the age of the child (from six months to twelve years), a mouth gag and scissors. The tubes are made of hard rubber, each tube fitting on a metal obturator that fits into the introducer.

When intubation is to be performed, the child is placed in a mummy sheet (a prone position is best) and the mouth gag is introduced on the left side of the mouth by an assistant. The operator, standing to the child's right and holding the introducer in his right hand, passes his left index finger

into the mouth until the epiglottis is felt; this he pulls forward to the base of the tongue, thus leaving the aperture of the larynx open. Keeping the index finger as much to the left as possible, he passes the introducer (on which has been fitted the proper tube with a string attached) backward along the median line until the clink of the glottis is reached. The introduction of the tube is then started by gradually elevating the handle of the introducer. Finally as the tube has entered well into the larynx the obturator is removed and, with the introducer, is withdrawn, and introduction of the tube is completed by the left index finger. The string is then cut and removed.

After a period of four to five days the tube is extracted by extubation, the method being essentially the same as in intubation, except that, as the tube is now in place, after fixation of the epiglottis the beak of the extractor is introduced into the lumen of the tube, and the latter removed.

The operation is apparently most simple. The novice, however, will find it is not so easy as it looks. Before intubation is attempted on the living subject the technique should be perfected on the cadaver.

There is no operation that can be performed so quickly that gives more startling and marvellous results than this. From a condition of slight or complete asphyxia the child is brought back in a few moments to a state of perfect comfort with all stenotic symptoms gone.

It may happen, of course, that if the tube is introduced into the esophagus the obstructive symptoms will not be relieved, and the tube can be felt to move freely from side to side, as well as upward and downward. It should then be removed and another attempt at insertion made.

After the intubation tube is in place it may remain so until extubation is performed or certain accidents may happen. It may be (1) coughed up into the mouth and either swallowed or expelled into the bed or on the floor. (2) It may be blocked by membrane. (3) It may be coughed up into the nasopharynx. (4) As rarely happens, it may be aspirated into the tracheal bifurcation. All these complications have certain symptoms and require obvious measures to overcome them, but space will not permit discussion of these at this time.

Certain precautions may be mentioned relative to intubation. One should never use force, and should endeavor to avoid all but the slightest bleeding. Care should be taken not to exhaust the child by too prolonged and oft-repeated attempts. The proper-sized intubation tube for the age of the child must be used. The string should never be cut until the operator is convinced that the tube is in place and the child is relieved.

Intubated patients may require special feeding methods. As a rule they may be fed in the usual way. For certain patients who cough the tube up with dangerous frequency, there is a special non-cough-up tube with a bulbous lower extremity that holds the tube firmly in place. There is also a special set of large-caliber tubes for adult patients, and there are dilating tubes of various sizes for use in patients with chronic laryngeal stenosis following diphtheritic laryngitis.

One of the greatest dangers of intubation is the frequency with which bronchopneumonia follows, especially in hospital centers. This will be referred to again.

(*F*) Tracheotomy. In the United States tracheotomy is used only in cases of laryngeal diphtheria when intubation, owing to the lack of technical skill, cannot be performed, or when the introduction of an O'Dwyer tube does not relieve the obstruction. It is hence a method of second choice or of last resort, and, as is to be expected under such conditions, the results are extremely poor. It is rare in our experience at the hospital, that a patient tracheotomized for relief of tracheobronchial membrane, after intubation has failed, recovers. Probably such cases in the future will be more and more treated only by suction, and little, if at all, by tracheotomy.

TREATMENT OF TRACHEOBRONCHIAL DIPHTHERIA

Such patients require very large doses of antitoxin both intravenous and intramuscular, and indeed the doses may need to be repeated for several consecutive days. One thing that must not be forgotten is the extent of the area of the membrane which may exist in the tracheobraonchial tree in well-advanced cases of this type, as compared to the smaller area of involvement in pharyngeal diphtheria. The difference

in the lymph supply in the two areas must also be comparatively considered. It is for these reasons, in my opinion, that very large doses of antitoxin are needed.

Intubation may be needed in such patients for membrane, edema or laryngeal spasm, but the child is usually not relieved at all, or only partially. Laryngoscopy with suction should therefore be employed frequently and as indicated by the patient's condition. Strychnine, spiritus frumenti, adrenalin, and small doses of morphine are usually urgently required.

Dr. Lynah used, in some of these cases, a very long, specially constructed intubation tube which he introduced into the trachea by direct bronchoscopy and laryngoscopy.

Complications of Diphtheria

These are due to two main causes: (1) The invasion of the body tissues by the microorganisms of secondary infection causing otitis media, bronchopneumonia, etc., (2) The action of the toxins on various tissues (heart, kidneys, nervous system, etc.) producing visceral syndromes or insufficiencies.

1. In the first group the main, and by far the most important, complication is bronchopneumonia. Otitis media occurs, but is an uncommon complication, limited usually to the septic forms of the disease. A diphtheritic otitis media is seen not infrequently in the course of other contagious diseases, especially measles.

Cervical adenitis as a complication, occurring after the subsidence of the angina, is exceptional. To see a suppurative process supervene at this time is rare.

BRONCHOPNEUMONIA. This is one of the most dreaded complications of diphtheria. It occurs mostly in the laryngeal, tracheobronchial and severe septic pharyngeal forms of the disease. When it supervenes within twenty-four hours of operation (intubation or tracheotomy) it is almost invariably fatal. In children under three years, especially in hospital centers, it is very apt to develop after operative intervention (excepting suction). Many times, no doubt, the lungs are involved when intubation is performed, but the signs of laryngeal stenosis mask the pneumonic symptoms.

The disease usually appears from the third to the sixth day after the invasion of the larynx. Ordinarily it is the elevation of the temperature, the dilatation of the alae nasi, and the cyanosis, rather than the physical signs, which call forth its recognition. At first there are signs of a diffuse bronchitis, but as the disease progresses, consolidated areas may be made out in various parts of the lungs. Coarse and subcrepitant râles are heard over a considerable part of the chest, especially if an intubation tube is in the larynx.

The disease appears in general to be due to an infection with a streptococcus, or less commonly a pneumococcus, originating from a buccal focus. The Klebs-Loeffler bacillus prepares the way for this invasion both in a mechanical manner and by lowering the tissue resistance. It is often found associated in the pneumonic area with the streptococcus.

Lobar pneumonia in diphtheria is exceptional. Pleurisy with effusion, either serous or purulent, is a very unusual complication in all except the septic fatal cases, where it may occasionally occur.

CARDIAC COMPLICATIONS. So frequent are cardiac symptoms in diphtheria that certain of them may really be classed under the symptomatology of the disease. More especially is this the case where these symptoms originate during the very acute stage, as in the "secondary syndrome of malignant diphtheria" previously mentioned, seen to supervene shortly after the membrane has disappeared from the throat. The same is true of the definite signs of the effects of the toxins on the cardiovascular system noted in those patients who die of polymicrobic diphtheria before the membrane has even disappeared from the pharynx.

Besides such cases, however, there is a larger group of patients who have passed through the acute attack into an apparently safe convalescence, but who still present symptoms of involvement of the cardiac musculature or its conducting mechanism. The symptoms, often rather latent, easily recognized and found if sought for, too often escape detection. They may not then be noted until aggravated by an undue degree of activity or exertion which the patient has unfortunately been permitted to make.

Practically we should never forget that the heart is affected to some extent in all moderate as well as severe cases of diphtheria. The degree of involvement may be so slight in many of the moderate cases, as to be lacking in subjective and objective symptoms. But it is rare that severe and very severe pharyngeal cases, those with the polymicrobic form and with membrane covering more than both tonsils, do not show cardiac insufficiencies of some kind. It is always to be remembered that the involvement of the heart is in a direct relation to the severity of the preceding angina, the extent of membrane formation and the number of days the patient was ill before antitoxin was administered. Recognition of this principle helps in indicating the evident prophylaxis.

The cardiac troubles of diphtheria manifested in convalescence are usually in patients with a peculiar waxy paleness who remain weak and profoundly intoxicated. The onset of symptoms is often gradual. There is a modification of the pulse, which becomes small and compressible, as though empty. Blood-pressure observations show a progressive diminution in arterial tension. The apex sounds of the heart show a rather typical change in the character of the first sound, which becomes muffled, and in the second sound, which is divided into two parts. Later systolic murmurs at the mitral valve due to muscular insufficiency may occur, and signs of dilatation of the heart may supervene. Signs of interference with the conducting mechanism of the heart often develop in the way of phenomenal excitation or inhibition. Finally, a group of symptoms similar to those noted in the secondary syndrome of malignant diphtheria may appear at any time, and the patient succumb. It may be, however, that even symptoms which seem surely fatal are gradually ameliorated and the patient recovers.

Pathologically, the lesions are observed in all parts of the cardiac muscle, either in the region of the bundle of His or in the undifferentiated muscle. They have the characteristics of inflammatory phenomena, in that they may retrocede. The signs of involvement of the "non-differentiated" cardiac muscles are accompanied by the usual signs of myocarditis. The lesions of the bundle of His, according to their extent, produce either troubles of excitation or phenomena of

inhibition. In the first group are auricular and ventricular extrasystoles, paroxysmal tachycardia, etc., while in the second are partial or complete heart-block, or total arrhythmia by auricular fibrillation.

In certain cases, besides the cardiac muscular lesions, lesions of the cardiac nerve endings, of the intracardiac ganglion, of the cardiac plexuses and of the vago-spinal system are apparently concerned.

PROPHYLAXIS OF THE HEART COMPLICATIONS. This is one of the most important points in the treatment of diphtheria. It comprises in brief a careful protection of the heart in all cases in which it is expected that symptoms will supervene. This protection is best given by a suitable period in bed in the recumbent position. This period should be proportionate to the preceding angina. Thus moderate cases of diphtheria should remain in bed from three to four weeks, moderately severe cases from four to six weeks and very severe cases from six to eight weeks. They should then only be allowed up if (1) on examination the heart is found in fairly normal condition, (2) the response to mild exercises shows the proper cardiac function, (3) the blood pressure has reached and remained at a normal figure for several days.

The actual treatment of the cardiac insufficiencies that develop consists of absolute rest, a most careful light liquid or semi-liquid diet, and the use of certain cardiac sedatives or stimulants as they are needed. Morphine is the drug par excellence in these cases, given in small doses, but best by hypodermic injection. Atropine may be given in conjunction with it. Adrenalin chloride in fairly large and frequent doses by subcutaneous injection is often indicated, especially when the blood pressure is low. Digitalis is occasionally used in cardiac insufficiencies from dilatation. One must be careful that it does not excite vomiting.

POST-DIPHTHERITIC PARALYSIS

(*A*) Frequence—relation to the types of the disease and the seat of the angina—time of apparition, (*B*) Local and general forms—symptoms, (*C*) Other characteristics of the paralysis, (*D*) Pathogenesis and pathological anatomy (*E*) Treatment.

(*A*) Paralysis follows diphtheritic angina in an average of 10 to 15 per cent of cases. The paralysis usually follows the severe forms of the disease, such as the polymicrobic types. They are far more frequent after pharyngeal than after laryngeal diphtheria. Certain epidemics or outbreaks due to certain stains of Klebs-Loeffler bacilli are followed by a higher percentage of paralytic complication than others. The paralysis may appear as early as the fifth to the fifteenth day, or as late as the first or second month after the disease. It may be localized to one muscle group, or it may be generalized.

(*B*) There are two main forms of post-diphtheritic paralysis: (1) The localized or common form, (2) The generalized, the true multiple neuritis form.

1. The localized paralysis comprises the palatine and palatopharyngeal paralysis.

Palatine paralysis is by far the most frequent paralytic manifestation of diphtheria. The onset of the condition is usually accompanied by slight general symptoms, such as pallor, slight fever, malaise and change in the character of the pulse. But it is the local signs by which it is most often recognized; that is, regurgitation of liquids through the nose, and nasal intonation of the voice. On examination of the throat the palate is partly or completely immobile, pale, and the uvula may touch the base of the tongue. The palatine reflexes are diminished or abolished.

There are two distinct types of palatine paralysis, so far as evolution is concerned. The first, the early or precocious paralysis, supervenes in the first ten days of the disease often when the false membrane has not yet disappeared. Their prognosis is very grave; they indicate a rapid and profound intoxication often terminated in death. The other, the tardive, a late group, develop after the twentieth day when the patient is in full convalescence, and are benign, recovery occurring generally in three to four weeks.

Palato pharyngeal paralysis is accompanied by the same symptoms as palatine paralysis with the addition of difficulty in swallowing semi-solid and solid foods, which may be partial or total.

2. Generalized Paralysis. The paralysis of this polyneuritic form is far less common than palatine paralysis,

and supervenes late in the disease, usually about the fourth to sixth week, and sometimes later. Owing to this fact, if the initial angina has passed without recognition, the cause of this multiple neuritis may at first be overlooked. Velopalatine paralysis is often the first symptom noticed; then follows involvement of the limbs, usually first the lower limbs, then the upper. Oculary paralysis may then supervene, with paralysis of accommodation and perhaps with ptosis and strabismus. Finally, in the worst cases, the neck, back, abdomen, diaphragm and intercostal muscles may be affected.

The generalized paralysis is of a mixed type, both motor and sensory nerves being concerned. The troubles of sensation are especially of the subjective type. They consist in formication, numbness, tingling without very clear topographical distribution. The lower limbs are generally only partially paralyzed. In fact, complete paralysis of the upper or lower limbs is exceptional. Not infrequently the neuritis in the lower extremities is limited to the form of a "pseudo tabes" with abolition of the reflexes and motor incoordination. The duration of the general paralysis is variable; it may last for several months, or even, exceptionally, an entire year. But the rule is that the paralysis patients practically always recover, leaving no sign of muscular atrophy behind. No matter how profoundly altered the peripheral nerves may be, the neuromuscular integrity is again ultimately established.

Deep reflexes in the affected parts are usually absent as long as the disease lasts. The electrical reactions of the muscles are the same as in any other multiple neuritis, with the exception that reaction of degeneration is rare.

(*C*) Oculary paralysis in diphtheria may occur alone or be associated with the preceding. The most frequent sign is the paralysis of accommodation. The light reflex remains but the accommodation reflex disappears. It is an inverse of the sign of Argyll-Robertson, and is very characteristic of diphtheria, but may occur also in botulism and epidemic encephalitis. Accommodation paralysis may last as long as four to six months.

Patients with post-diphtheritic paralysis recover always, unless there is, in a vital part, such as the heart or the muscles of respiration, a disturbance of essential function. Occasionally patients with generalized paralysis are carried off thus in the course of the development of a syndrome aptly called "bulbar crisis." At times asphyxiation, mechanically induced by the aspiration of a piece of food into the air passages, produces sudden death. Or again, a deglutition pneumonia may follow the aspiration of food particles and lead to a fatal termination.

(*D*) Pathological Anatomy and Pathogenesis. Today it seems to be the general view, following the teaching of Babonneix, Gullain, Larouche and Walsh, that the local and the general diphtheritic paralysis arise in different ways. The local paralysis, according to this view, has its origin in an ascending lymphogenous toxic infection of the central nervous system occurring from the site of the initial angina. The essential pathological lesion here is central, and is situated in the glossopharyngeal-vagus accessory nuclear system. The general paralysis, including probably the ocular paralysis, is the result of the circulation of the toxin in the blood stream whence it gains access to the whole nervous system. Here the lesion is central and peripheral.

Cerebrospinal Fluid. In post-diphtheritic paralysis this is a clear limpid fluid, flowing under normal or slight hypertension. The cell count is normal lymphocytic in type, the sugar reaction shows a good reduction (3 plus), the albumin is slightly increased in 30 to 40 per cent of cases, and quantitatively amounts to 0.40 to 0.80 gms. per liter. The globulin reaction may also be slightly positive in a minor proportion of cases. The colloidal gold reaction occurs in the luetic zone, but, in some fluids, is prolonged slightly into the meningitic zone.

(*E*) Treatment. Without attempting to speak of the general care of the disease, so important in the severe cases, it may be said that the treatment comprises two things: the first is prolonged and adequate rest in bed and the other the administration of strychnine, by mouth in the milder cases, by injection and in larger doses in the severe cases.

Treatment of Diphtheria in General

(*A*) Local methods, (*B*) Specific.

Local measures of treatment should be limited to the use of mild antiseptics in the nose and throat.

Treatment comprises, above all things, the administration of diphtheria antitoxin in adequate dosage and early in the disease. Diphtheria is one of the few diseases for which we have a specific therapy, and it seems criminal that cases should be lost today from failure to administer antitoxin on time.

The dosage of antitoxin is proportionate slightly to the age and weight of the child, but mostly to the duration and severity of the disease.

DOSAGE OF DIPHTHERIA ANTITOXIN

(*A*) Pharyngeal diphtheria—intramuscular route.

	Mild	Moderate	Severe	Very Severe
Young children	5,000–8,000	8,000–15,000	20,000–30,000	30,000–40,00
Older children and adults...	8,000–10,000	10,000–20,000	20,000–35,000	35,000–50,000 or 70,000

(*B*) Nasal diphtheria—Mild 5000–10,000; severe 20,000–30,000

(*C*) Laryngeal diphtheria—15,000–25,000 units

(*D*) Tracheobronchial diphtheria—30,000—50,000 (repetition of part dose)

ROUTES OF ADMINISTRATION OF ANTITOXIN

(*A*) Considerations

1. Subcutaneous—Attains maximum in blood only in 36–48 hrs.
2. Intramuscular (buttocks)—Attains maximum in blood in 8–12 hrs. (remaining thus 48 hrs.).
3. Intravenous—Attains maximum in blood immediately (but ⅔ disappears in 24 hrs.).

(*B*) Conclusions

1. Intramuscular (gluteal muscles) best for all mild, moderate cases

2. Intravenous best for all severe and very severe cases, and for laryngeal and tracheobronchial types.
3. Best to supplement all intravenous injections by intramuscular injections

(*C*) Intravenous administration
1. The special refined antitoxin must be used
2. Aseptic technique and general precautions for intravenous injections
3. Dosage from 10,000 to 15,000 units; occasionally higher
4. Avoid its use in sensitized patients

PROPHYLAXIS OF DIPHTHERIA INCLUDES

(*A*) Prevention of dissemination by quarantine regulations
(*B*) Active immunization of all susceptibles by toxin antitoxin, susceptibility being determined by Schick reaction
(*C*) Passive immunization of all susceptibles closely exposed, by antitoxin (1000–1500 units)
(*A*) Prevention of dissemination: includes control of
1. Dissemination by nose, throat secretions by direct (common), indirect contact (less common), from the sick to well
2. Carriers
3. Missed cases, abortive and atypical cases
4. Carrying out of quarantine regulations

(*B*) Schick test
1. Material used
2. Reaction

(*C*) Toxin antitoxin
1. Amount injected
2. Frequence
3. Time of development of immunity
4. Duration

The technique of the Schick test and the details of administration of toxin antitoxin are so familiar to all, following the extensive publications of Dr. Park and Dr. Zingher, that there is no necessity of more than reference to them, and to other prophylactic measures as outlined above.

EPIDEMIC ENCEPHALITIS*

FREDERICK TILNEY, M.D.

DURING the past decade three devastating epidemics have swept over this country. Strangely enough, two of them have involved the nervous system primarily. With the first of these everyone is familiar. It has passed into history although some of its dire effects are still to be seen in and about the town. This epidemic of acute anterior poliomyelitis occurred in 1916. It claimed some 26,000 victims throughout the United States, nearly 9000 of whom were in New York City alone.

In 1918 another epidemic involving the nervous system appeared in this country following the demobilization of the troops after the great war. It has been in process ever since, i.e., epidemic encephalitis.

Communication with the health departments of many cities in the Union concerning this epidemic and also with the Federal health department, leads one to conclude that a conservative estimate would place the number of these victims in our country alone at about 200,000. It has been a widespread disease during the past several years. Moreover, during the past year, it seems to have been in a phase of recrudescence, and the number of cases which have been seen and recognized is somewhat more than at any other period since the beginning of the epidemic in 1918.

In order to present as concisely and tersely as possible the details concerning this disease—its natural history, its lines of spread as well as its symptomatology, a brief outline of its major features has been prepared with a view to giving a synopsis of facts rather than a more formal discussion.

It has frequently been asked: Is this an entirely new disease? Apparently one would be inclined to decide in the negative after consulting medical literature, some references to which are given below as a historical retrospect:

*This lecture was delivered on May 4, 1923.

ANCIENT:	PHRENITIS OR CAUSUS OF ARETAEUS AND GALEN?
1521:	MODORRA OR MORDORRILLA. *Lisbon.* DE SOUSA.
1561:	PESTILENCE SOPOREUSE. *Italy.* AMATUS. MAL MAZZUCO.
1695:	LETHARGY WITH OCULAR PARALYSIS. *Germany.* ALBRECHT.
1712:	SCHLAFKRANKHEIT. *Tubingen.* CAMERARIUS.
1890:	NONA (MALATTIA DE LA NONA). *Italy.* LONGUET.
1892:	LA GRIPPE CEREBRALE. PHAHL.
1917:	ENCEPHALITIS LETHARGICA. VON ECONOMO.
1918:	EPIDEMIC ENCEPHALITIS. HALL-WILSON.
1920:	*ENCEPHALITIS LETHARGICA*—Admitted as an Independent Disease to the Ninth Edition of Osler's Principles and Practice of Medicine.

From such references as these this malady appears to be an ancient disease, known even to Galen among the ancients and *phrenitis* and as *causus* by Aretaeus. It was also known in the Renaissance, and described most carefully by De Sousa in Italy, where it was referred to as the *pestilence soporeuse* or *mal mazzuco.* In Germany, in 1695, it was well described as lethargy with ocular paralysis. Then came the well-known incident in 1712, when a whole town apparently was overcome by a sleeping sickness which merited the term *schlafkrankheit.* In 1890 it was known in northern Italy as *nona* (*malattia de la nona*) and described by Longuet. In 1892 the influenza epidemic went around the world and this disease was spoken of by Phahl as *la grippe cerebrale.* In 1917 the disease was described by von Economo in Vienna as Encephalitis Lethargica.This designation was attacked by two Englishmen, Hall and Wilson, on the ground that it was not the encephalitis that was lethargic, but the patients. They accordingly suggested the term epidemic encephalitis, which perhaps is a better name, although not entirely free from criticism. Finally, in 1920, the term *encephalitis lethargica* was sanctioned by its appearance in the Ninth Edition of Osler's "Principles and Practice of Medicine." We pass next in our summary to another important question, namely, possible mutuality in relation of lethargic encephalitis to certain other epidemic diseases.

POSSIBLE MUTUALITY IN RELATION OF LETHARGIC ENCEPHALITIS TO CERTAIN OTHER EPIDEMIC DISEASES

It appears from the facts thus far obtained that influenza and encephalitis are not the same disease and furthermore encephalitis is not a nervous complication of influenza. Nor for that matter is influenza a common predisposing cause of encephalitis. A review of some recent epidemics is tabulated below and furnishes the basis for the foregoing conclusions concerning certain mutualities in the disease:

Encephalitis	Date	Time Relation	Influenza
Vienna Epidemic	1917	Preceded by Months	Pandemic of 1917
Winnipeg Epidemic	1919	Followed 12 Months after	Epidemic 1918
France, England and United States Epidemics	1918 1919 1920	Coincident with	Epidemics of Same Years
Switzerland Epidemic	1920	Coincident with	Epidemic 1920
Switzerland No Cases	1918–1919	Not Related	Pandemic 1918–1919

Acute Anterior Poliomyelitis Clinically and Pathologically Seems to be a Different Disease While Epidemic Hiccough May Be Closely Related to, If Not Identical with, Lethargic Encephalitis.

Considering next the spread of this disease, it appears to have started in Vienna, in 1917, and followed the westward course of the troops through northern Italy, into Paris, and thence to London. It appeared almost simultaneously in Paris and London, in 1917, probably because of the intercommunication across the English Channel in the movement of the troops. It appeared somewhat later in Spain, and later still in Germany and Sweden. In the fall of 1918, in New York City, we first began to see a large number of cases of a mysterious sort. In them stupor or profound and protracted somnolence, combined with paraly-

sis of the cranial nerves were the most important features. In its spread it extended a little to the south, Philadelphia and Washington having very few cases, and then it veered to the north toward Boston. In the winter of 1918 it appeared for the first time in Chicago and then invaded Canada, where a pronounced epidemic was described by Boyd in Winnipeg. Crossing the continent and still going westward, it appeared in the winter of 1920 in San Francisco; and since then it has followed these general lines of transit in its spread across this country, confining itself more to a zone midway between the north and the south.

Another interesting question concerning the disease is its possible relation to influenza. Present statistics seem to show that there is no intimate relation of this kind. The epidemics cited above indicate that in Vienna encephalitis was preceded by several months by influenza, while in Winnipeg it followed twelve months after the epidemic of influenza. The figures obtained from France, England and the United States are not so conclusive, but those from Switzerland appear to be more definite, and indicate that there were no cases of encephalitis, no influenza epidemic on the Continent or in this country at that time.

There is one condition that has occurred frequently, namely, epidemic hiccough, which seems more closely related to epidemic encephalitis, if not actually identical with it.

Concerning the period of incubation the following tabulation gives the prevailing views:

INCUBATION PERIOD

Strauss and Loewe, *New York*	9 to 40 Days
British Ministry of Health	4 to 33 Days
Derby Statistics	26 Days
Boyd, *Winnipeg*	21 to 40 Days
Sabatini, *Italy*	20 Days

The period of incubation thus shows considerable variation as estimated by a number of different observers. We are inclined to accept the view of Strauss and Loewe, whose work we think is among the best. They give the period of incubation as nine to forty days, and that seems to strike a fair

average. The British Ministry of Health gives it as a little less—four to thirty-three days. The Derby Statistics give it as twenty-six days, Boyd in Winnipeg, twenty-one to forty days, and Sabatini in Italy, twenty days; so somewhere between four and forty days seems to be the average period of incubation after exposure.

Etiologically there is also a variety of opinions concerning this disease as shown in the following tabulation:

BACTERIOLOGICAL EXPLANATIONS OFFERED

1917	Wiessner	Filter Passing Virus—Pleomorphic Diplo-Streptococcus
1918	McIntosh	Gram Positive—Motile Nonproteolytic Bacillus (Like Vibrion Septique—Pasteur)
1919	Strauss, Hirshfeld and Loewe	Filter Passing Virus from Brain, Nasal Washings, Spinal Fluid and Blood. Filterable Organism
1919	Thalheimer	Same Results as Strauss, Hirshfeld and Loewe
1920	Levaditi and Harvier	Filter Passing Virus Effective in Rabbit and Guinea Pig—Not for Monkey
1920	Doerr	By Injecting Virus of Herpes Labialis Produced Encephalitis in Rabbits
1922	Levaditi and Harvier	Causal Organism Contained in Filter Passing Virus Existing in Four Forms: 1. Attenuated in Normal Human Saliva 2. Virulent in Vesicles of Herpes 3. Virulent in Encephalitic Carriers 4. Most Virulent in Central Nervous System of Frank Cases
1922	Rosenow	"Green Producing Streptococcus" from Infected Teeth, Nasal Washing Filtrate, Blood, Emulsion Base of Brain and Filtrate of Base of Brain

Many explanations have been offered concerning the bacteriology. Wiessner found the cause of the disease to be a

filter passing virus—pleomorphic diplo-streptococcus. McIntosh found a gram positive,—motile nonproteolytic bacillus. Strauss, Hirshfeld and Loewe found an organism, which has been much talked about, in washings from the nasal cavity, spinal fluid and blood. Thalheimer, of Milwaukee, confirmed the findings of Strauss, Hirshfeld and Loewe. Levaditi and Harvier practically came to the same conclusions. Doerr was able to reproduce the disease in rabbits. Some further work by Levaditi and Harvier showed that the organism may exist in several different forms.

Recently Rosenow, of the Mayo Clinic, at the Academy of Medicine, New York, gave it as his opinion that the organism producing this disease is a pleomorphic streptococcus, a streptococcus which changes its form. This seems to agree with the ideas of Levaditi and Harvier. The organism of Rosenow is a "green producing streptococcus" which he gets from infected teeth, nasal washings, emulsions from the base of the brain and filtrates of the base of the brain. He described a very interesting experiment involving 3 cases from the Neurological Institute. One was the case of a colonel who, for three months had a severe type of delirium and twitching of the neck muscles. Rosenow took washings from the nasal cavity, injected them into a rabbit and produced the same phenomena in the animal. The second case was one of Dr. Timme's, that of a man with a highly delirious form of affection, with difficulty in swallowing and difficulty in speech. Rosenow injected washings from this man into a rabbit, producing a peculiar delirious state in the animal, and causing its death in forty-eight hours. Twelve hours later the patient himself died. The third, a case of Dr. Dana's, had a peculiar tic of the neck muscles. From this case Rosenow injected an animal and produced the same clinical appearances in the animal. Rosenow argues that this pleomorphic organism of his is so peculiar in its morphology that certain strains of it will produce the definite types with the clinical appearances of this disease with which we are familiar. This is presented merely as a fact, with no comment on it except that it is very difficult to interpret the clinical appearances in rabbits. Still

it does not seem at all certain that the contention of Rosenow is a valid one in explanation of this disease.

GENERAL SYMPTOMS

Fever	About 14% Moderate Pyrexia 72%	Protracted Pyrexia 13% Hyperpyrexia 1%
Circulatory	Independent Tachycardia and Bradycardia } Unusual	Hypertension Common Blood Usually Normal
Respiratory	Usually Follows Pulse and Temperature Except Where Embarrassed by Central Lesions Producing Dyspnea, Hiccough, Abdominal Myoclonus or Cheyne-Stokes Breathing	
Digestive	Tongue Usually Coated, Dry and Cracked Marked Constipation in Most Cases Nausea and Anorexia Frequent	
Cutaneous	Eruptions of Many Varieties in About 9% of Cases	

As to the general symptoms of the disease: The first is fever. This has been a matter of much debate. There is no fever in 14 per cent of the cases. There is a moderate pyrexia in 72 per cent, a protracted pyrexia running through months in 13 per cent, and a hyperpyrexia with remissions in 1 per cent of the cases.

The circulatory symptoms are not particularly marked. Sometimes there is a tachycardia, and sometimes a slowness of the heart and often fluctuations in the heart rate. Hypertension is common, although the blood picture itself is usually normal.

Respiratory changes follow the pulse and temperature and are embarrassed only when the central nervous system, particularly the bulb, is affected, with resultant dyspnea, hiccough, abdominal myoclonus or Cheyne-Stokes breathing.

As to digestive symptoms, the tongue is usually heavily coated, cracked and dry, in some cases looking not unlike a typhoid tongue. Marked constipation is present in most instances. Nausea and anorexia are of frequent occurrence.

What is not generally recognized in this disease is the fact that 9 per cent of the cases present various sorts of affections of the skin which include almost every form of cutaneous eruption.

These are the general or systemic symptoms.

CHARACTER OF ONSET

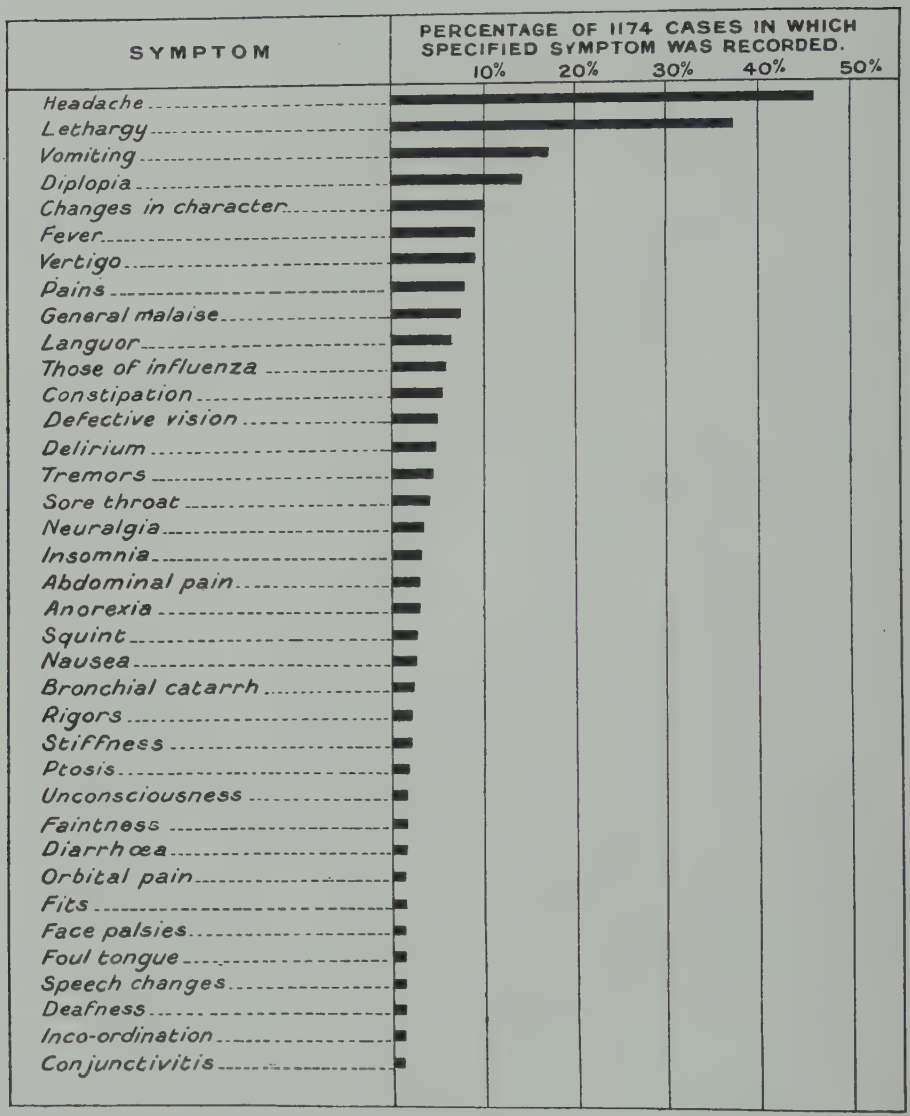
Acute Onset 6%–12%	Gradual Onset 88%–94%
Chills and Pyrexia	Sudden Visual Defect
Sudden Coma	Hemianopsia
Apoplectiform Symptoms	Diplopia
Hemiplegia	General Malaise
Aphasia	Headache
Sudden Paralysis, Legs or Arms	Generalized Pains
Abdominal Twitchings	Pains in Back and Arms
Catalepsy	Pains in Legs
Vomiting	Asthenia
Giddiness and Vomiting	Increasing Insomnia
General Convulsion	Increasing Restlessness
Focal Spasm	Increasing Somnolence
Sudden Delirium	Recurrent Visual Defects
Sudden Exaltation or Mild Mania	Vertigo

The character of the onset is perplexing and often quite indefinite. Many cases begin with a rather gradual and insidious onset, the disease being ushered in by any one of the above symptoms, which in so far as may be, are arranged in order of frequency. The onset may also be sudden and acute.

The initial symptom in order of frequency is indicated in the following chart:

This chart gives the estimated frequency of the initial symptom of this disease in 1174 cases. Headache stands at the head of the list, with nearly 50 per cent, lethargy coming next with about 40 per cent, vomiting next with about 20 per cent, and diplopia fourth. This last symptom now seems to stand somewhat lower in the scale, although in the first part of the epidemic it seemed to stand first.

These symptoms show a wide range of disordered function in the central nervous system.


SYMPTOM
PERCENTAGE OF 1174 CASES IN WHICH SPECIFIED SYMPTOM WAS RECORDED.
10%
20%
30%
40%
50%
Headache
Lethargy
Vomiting
Diplopia
Changes in character
Fever
Vertigo
Pains
General malaise
Languor
Those of influenza
Constipation
Defective vision
Delirium
Tremors
Sore throat
Neuralgia
Insomnia
Abdominal pain
Anorexia
Squint
Nausea
Bronchial catarrh
Rigors
Stiffness
Ptosis
Unconsciousness
Faintness
Diarrhœa
Orbital pain
Fits
Face palsies
Foul tongue
Speech changes
Deafness
Inco-ordination
Conjunctivitis

Headache, the most common symptom, is intense and usually protracted, involving the entire head.

PARTS OF NERVOUS SYSTEM INVOLVED AND RESULTING FOCAL SYMPTOMS

1. Oculomotor Nucleus
   - palsies of extrinsic and intrinsic eye muscles

2. Basal Ganglia—Including Striatum, Thalamus and Red Nucleus
   - paralysis agitans
   - athetosis
   - chorea
   - choreoathetosis
   - mobile spasm
   - cataleptic rigidity
   - myorhythmic movements
   - emotional imbalance

3. Pons Varolii
   - facial paralysis
   - abducens paralysis

4. Cerebral Cortex
   - delirium
   - hallucinosis
   - confusion and mania
   - behavior perversions
   - hypersomnia
   - insomnia
   - aphasia
   - anesthesia
   - convulsions
   - astereognosis

5. Spinal Cord
   - myoclonic spasms
   - flaccid paralysis

6. Dorsal Roots
   - radicular pains

7. Medulla Oblongata
   - vertigo and tinnitus
   - nausea and vomiting
   - dyspnea and tachycardia
   - dysphagia
   - respiratory and cardiac death

8. Internal Capsule
   - hemiplegia

9. Cerebellum
   - asynergia
   - nystagmus

10. Optic Nerve
    - amblyopia
    - optic neuritis
    - choked disc

11. Pituitary Body
    - froehlich's syndrome

12. Sympathetic System
    - sympathicotonia
    - basedow's syndrome
    - addison's syndrome

13. Dorsal Root Ganglia
    - herpes zoster

The tendency for certain parts of the nervous system to be more frequently affected than others is a most interesting consideration. The oculomotor involvement which is one of the most common, gives rise to palsies of the extrinsic and intrinsic muscles of the eye. This may be due to the fact that

the circulation here is an end-circulation, and some small branches of the blood vessels which leave the circle of Willis, almost at right angles, may perhaps provide an easy approach to the central nervous structure of the midbrain.

Next in order of frequency are the basal ganglia, giving rise to paralysis agitans phenomena, stiffness of the body, or a general rigidity of the musculature, so that the patient lies in immobile lethargy, perhaps for weeks at a time.

The Pons varolii is next in order. When this tabulation was originally made the cerebral cortex was placed in fourth position. It seems advisable to change that order now and place it second, because of the fact that most of the cases seen this year have presented some form of delirium, hallucinosis, confusion or mania, and, most remarkable of all, certain behavior perversions. These occur especially in children who act most peculiarly as a result of this disease. Parents bring children of this kind to the doctor with all sorts of theories as to their unusual misconduct. From their history it becomes apparent that there has been an inflammation in the brain undoubtedly affecting the cortex and giving rise to changes in behavior. Increased sleep also, is probably due to a cortical rather than a brain-stem involvement. Insomnia, aphasia, anesthesia, convulsions and the loss of proper sense of perception of objects by palpation (astereognosis) are symptoms also due to involvement of the cerebral cortex.

The spinal-cord form of the disease is well known and is especially common with its myoclonic twitchings, particularly of the diaphragm, and in the abdominal muscles. Furthermore, flaccid paralysis, closely resembling that of anterior poliomyelitis, has led to the belief that these two diseases are intimately allied.

The dorsal roots and the root ganglia are commonly the seat of the main involvement. Inflammation here gives rise to those vicious pains which last for weeks and months, for which no remedy seems availing.

The medulla oblongata, when involved by the inflammatory process, causes symptoms of grave portent. This is especially true when there are signs pointing to cardiac involvement. Such cases will in all probability terminate fatally.

The internal capsule has also been found to be involved, giving rise to hemiplegia.

The cerebellum may also be affected, causing asynergia and nystagmus.

Optic-nerve involvement may be a symptom, even giving rise to choked disc, which closely resembles the choked disc of brain tumor. If the choked disc in encephalitis is carefully studied, and also the retina, it will be found that the vessels show bulbous dilatations where the plasma cells are packed in around the perivascular lymph spaces very much as they are in the brain. It must be remembered that the eye is really an evaginated part of the brain, so the involvement here would be very similar to that in the actual brain tissue.

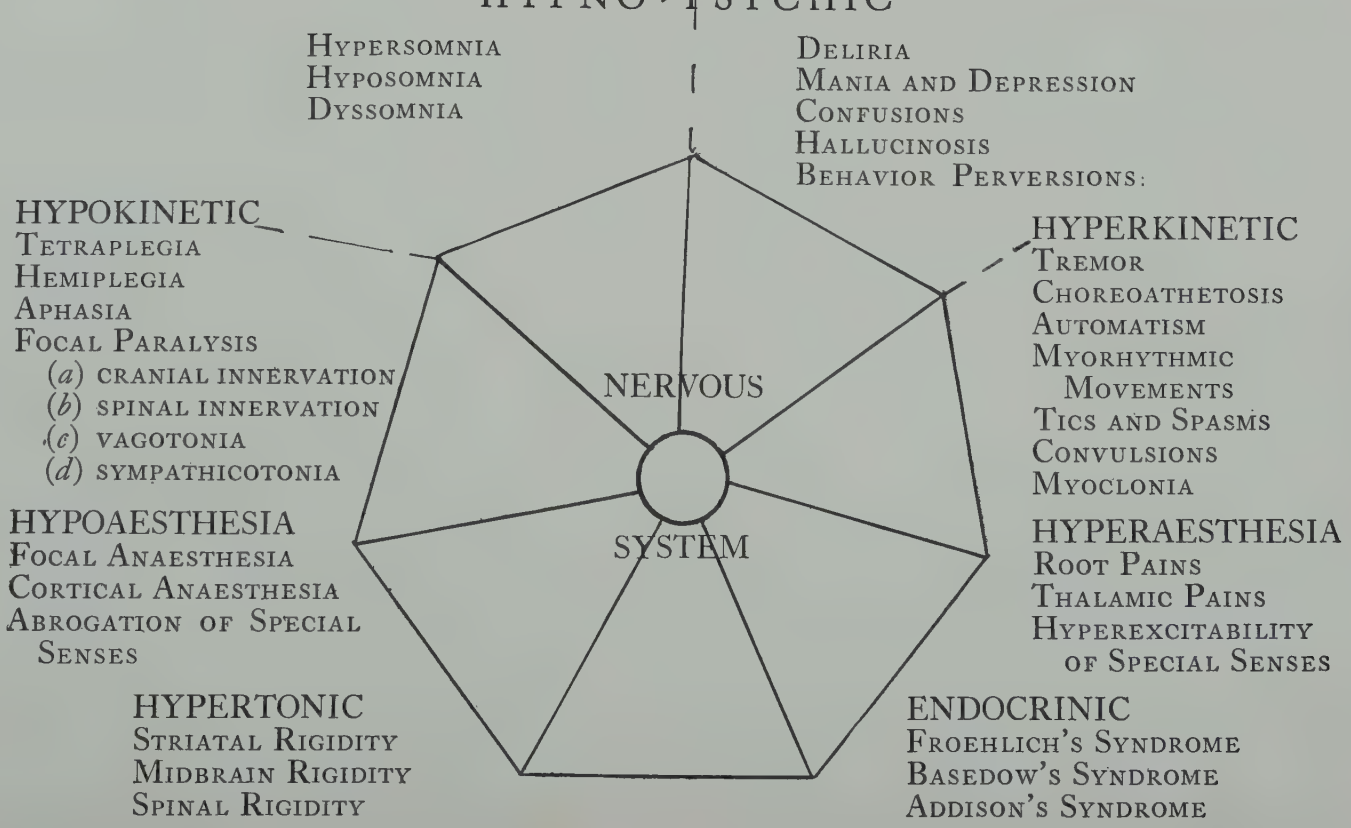
A great many patients gain weight during the course of the disease, become very heavy, and, in fact, show all the symptoms of the pituitary syndrome of Froehlich. There are, on the other hand, some that lose weight rapidly, with tachycardia, fine tremor and exophthalmos—all the symptoms of Basedow's disease. Any attempt to remove the thyroid in such cases almost invariably results fatally, because it is not the thyroid which is primarily affected, but the sympathetic nervous system.

There is also an Addisonian form of the disease in which the body first presents an intense scarlet eruption which lasts three or four days, accompanied by high temperature. Following this there is a gradual bronzing of the body, with a fall in the blood sugar, temperature and blood pressure. Death usually supervenes in a few days.

The dorsal root ganglia may be affected in connection with the cervical, lumbar and thoracic nerves, giving rise to herpes zoster, i.e., an actual posterior poliomyelitis.

In considering the types of this disease, and in grouping the cases clinically, it is almost impossible to make clinical divisions affirming that one case represents one group and one another, because it is the central nervous system which is suffering from the infection, and any part of it may be involved, or several parts simultaneously. Such being the case, there results a combination of symptoms. Perhaps some one group of symptoms may predominate, and this may lead to a justifiable distinction of types.

TYPES OF ENCEPHALITIS LETHARGICA



In the early part of the epidemic the hypnopsychic group of cases was recognized, in which the patient either slept all day or was awake all night or was very irregular in sleeping. There was also the lethargic group. Combined with this there were many cases of delirium, mania and depression, confusions, hallucinosis and behavior perversions. This variety constitutes one large group, but it might easily blend with others, and give symptoms combining other symptomatic possibilities. It is difficult, therefore, to make any distinct grouping at the present time. We do recognize paralysis, aphasia, reduced sensibility in general and special senses, and increase in tone of the body muscles.

PATHOLOGICAL ANATOMY

Concerning the pathology of the disease, it has almost universally been found that the lesions are largely confined to the blood-vessel spaces, i.e., the perivascular lymph spaces. In some areas there are actual hemorrhages, but where no hemorrhages occur, the perivascular spaces are packed with round cells and with plasma cells. The important feature about this pathological change lies in this fact: Either these ultra-microscopic organisms are retained in these rests for a long time, or else are contained in the cells collected in the perivascular lymph spaces. Anything that tends to elevate the blood pressure, even when the patient has apparently recovered and gone back to work, will have the tendency to scatter these cells again in the brain tissue and produce a reinfection. This is apparently the explanation of the remarkable frequency with which relapses occur, often after weeks or months, and indeed after several years have passed since the initial attack. The lesions are almost invariably of the irritative type. They are unlike those of anterior poliomyelitis, which are essentially destructive. It is probable that many of these peculiar rhythmic movements, marked increase in the tone of the body and tremors, are due to the fact that the pathology here favors irritative stimulation of various parts of the nervous system, in contradistinction to anterior poliomyelitis,which destroys and produces paralysis.

The small blood vessels crowded with these plasma cells, as well as many small lymph cells in the perivascular lymph spaces, with some reactive resistance in the neuroglia, seem to point to a definite reaction which protects the structure of the brain from further extension of the lesions.

The nerve cells themselves do not actually escape. They certainly appear to have an abnormal appearance. They are, however, not in a state indicative of a destructive process. What they do present is a condition of cloudy swelling, but there is no actual chromatolysis nor gathering of satellite cells around the nerve cells. The picture is quite different from anterior poliomyelitis, which is preeminently of the destructive type.

The important practical feature in the study of this disease is a careful diagnosis. This disease bears many striking resemblances to a number of other disorders. For example, when cases of encephalitis first began to appear, the writer was called to the Brooklyn Hospital, where in three adjoining beds of the same ward were 3 cases, all suspected to be sleeping sickness. One of these patients had already gained newspaper notoriety and had been written up very extensively as a case of "sleeping sickness." His next-door neighbor presented deep somnolence, ocular palsies, a stiff neck, and gave a history which was perfectly consistent with that of encephalitis. The course of the disease, however, was a little different in this case because he rapidly ran on to a more and more stuporous state and finally died in about two weeks. His headache, deep sleep, mental and nervous symptoms were all perfectly characteristic of epidemic encephalitis, but the spinal fluid showed salient differences. A great number of cells was found, something like 2000 lymphocytes, and the tuberculous organism was recovered from the spinal fluid. The blood was negative. Here there was a possibility of confusing a tuberculous meningitis with epidemic encephalitis. The case of his next-door neighbor, who had all the symptoms clinically of sleeping sickness and was reported as such, upon examination of the spinal fluid showed a 4-plus Wassermann reaction which in itself, made the distinction. The serology in these three cases which seemed so similar clinically furnished the necessary differential data.

In the several forms of meningitis, meningococcus, pneumococcus, streptococcus, epidemic meningitis, the symptomatic correspondence may be striking in all of the clinical details, except that the motor symptoms are very much more pronounced. The spinal fluid, of course, will reveal the character of the infecting organism, be it streptococcus or pneumococcus, so that differential diagnosis may be established in this way. It is usually not a difficult differential diagnosis to make in this disease, but the convergence of types may be such as to confuse the points particularly between anterior poliomyelitis and lethargic encephalitis.

Multiple sclerosis is another condition in which the history will establish the main point of differentiation, but perhaps the most important thing to distinguish between these two conditions, lethargic encephalitis and multiple sclerosis, is the alteration in the optic discs. The most outstanding feature here is the bitemporal pallor which is prominent in multiple sclerosis No difficulty should be experienced as a rule between these two conditions.

In some cases typhoid fever is sufficiently like epidemic encephalitis to cause perplexity. There was a patient whom the writer followed for five weeks with Dr. Evan Evans and Dr. Rhinethaler, of New York City. It was thought up to the end of the fifth week that she had typhoid fever, but she then developed a diplopia, a double ptosis and a bilateral facial paralysis, which was so unlike typhoid fever that the diagnosis was changed to epidemic encephalitis. About a week later she died and the autopsy showed that the last diagnosis was correct. As a rule the main point is the detection of the organism in the blood or the presence of a Widal reaction. These usually suffice in making the distinction.

What has confused a good many observers is the differential diagnosis between a mild case of epidemic lethargic encephalitis and ophthalmic migraine. Such a case was that of a young woman who gave a history of repeated attacks of ophthalmic migraine. She had hemianopsia and double vision. The history showed that the attack was not a typical ophthalmic migraine and that she was actually suffering from lethargic encephalitis. She began to run a low temperature and also had double vision, which continued long

beyond the period that it should on the basis of ophthalmic migraine; so it was concluded that she had lethargic encephalitis. She has been sleeping peacefully since, then; now, a period of three and a half weeks. So the history in this case seems to show the chief point of distinction between this relatively simple condition and epidemic lethargic encephalitis.

Circumscribed lesions in the brain, such as those duct's hemorrhage, thrombosis and embolism, often occasion some diagnostic difficulty, but the history, the mental and motor symptoms of the patient together with the history of development, the general systemic condition, should establish the chief differential features. One of the most difficult differential diagnoses to make is that between epidemic encephalitis and brain tumor. The history, if carefully taken, will give the distinguishing features between the encephalitic process and brain tumor. One point deserving of attention here is the retina. Choked disc, of course, in brain tumor, is almost a *sine qua non* to the diagnosis as we look at it now; but epidemic encephalitis *may* also have choked disc. A careful survey of the retina, especially the vessels in the disc, will often reveal a bulbous dilatation which seems to occur more commonly in epidemic encephalitis than in any other disease, and is one of the important differential points between this condition and brain tumor.

A very interesting case was reported by the late Dr. Henry G. Webster, in which the patient was supposed to be suffering from lethargic encephalitis. That patient had her tonsils removed about six months before, and then ran a slight temperature and began to sleep. She suffered from very severe headache. The sleep, the headache and the disturbance in the eye muscles led to the diagnosis of epidemic encephalitis. This patient, on *x*-ray examination, showed a lung abscess, and lung abscess following removal of a tonsil occurs, according to some observers, in about 10 per cent of cases; so the history there was perfectly clear. It was a logical sequence,—removal of the tonsils, lung abscess, metastatic abscess of the brain revealed not only in the history but by examination of the blood which showed a marked leucocytosis, and also by a study of the cells in the spinal

fluid which showed a definite inflammatory reaction; and the regular temperature of a chronic brain abscess, just a little bit above or a little below the normal line from day to day. So this case seems to be a striking example of the difference between epidemic encephalitis brain abscess, on the basis of the history and the findings in the spinal fluid and the blood.

The question always arises: What can we offer in the prognosis of this disease?

PROGNOSIS

Mortality—High and Low Estimates	12–48%
Mortality—Average Estimate	25%
Recovery with Residuals (Average)	63%
Complete Recovery	12%
Progressive Extensions in Acute Period	60%
Relapses Several Weeks After Apparent Recovery	20%
Recrudescence Several Months or Years After Recovery	15%
Uninterrupted Recovery	5%
Average Duration of Acute Period	6 to 8 Weeks

It is to be noted that high and low estimates of mortality vary from 12 to 48 per cent; average estimates of mortality, 25 per cent; recovery with residuals, 63 per cent; complete recovery, 12 per cent; progressive extensions in the acute period, 60 per cent; relapses several weeks after apparent recovery, 20 per cent; recrudescence several months or years after recovery, 15 per cent; uninterrupted recovery in 5 per cent of cases; average duration of the acute period of the disease, six to eight weeks.

The average duration of the entire course of the disease cannot be stated because some patients who had this disease in 1918 are still suffering from it.

MOST COMMON RESIDUAL DISTURBANCES

Psychic Disorders	Permanent Cranial Nerve Palsies
Emotional Instability	Recurrent Cranial Nerve Palsies
Personality Perversions	Choreiform Movements
Insomnia	Spastic Paralysis
Recurrent Drowsiness	Flaccid Paralysis
Paralysis Agitans Syndrome	Convulsions
Other Tremors	Ataxia
Focal Myoclinic Spasms	Froehlich's Syndrome
Myorhythmic Movements	Basedow's Syndrome

Speaking of the treatment, one must be very guarded. There have been many exaggerated statements made concerning it. That there is still no specific treatment for the disease seems certain. Many kinds of sera have been tried. The non-specific horse-serum and foreign proteins generally, are dangerous. The Loewe and Strauss serum has been practically abandoned by those investigators. The immune serum which was used for a time, obtained from patients who had the disease, is now considered of no avail, either as a prophylactic or as an actual radical cure. Dr. Timme and Dr. Dana have been trying Rosenow's serum recently in New York, and at a recent meeting they spoke rather conservatively about its use. The writer tried it in 7 cases; in a very acute case with Dr. Luther Warren in which we gave some twenty-five intravenous injections and the patient went on without apparent benefit to a fatal termination at the end of about three weeks; in chronic cases in which no good results were obtained from it; also in some of the more acute cases at the Neurological Institute; and the conclusion is reached that recommendation for it must be very guarded. Perhaps in a case where everything else seems to be failing it might be tried.

Many drugs have been advocated and used to combat the symptomatic progress of the disease. Urotropin is highly recommended and used almost universally. In asthenic cases adrenal residue is one of the most helpful agents, 10 to 15 minims every two hours, depending on the blood sugar and the blood pressure. If the blood pressure is low the dose should be accordingly. Scopolamine and hyoscine have been

recommended, but they must be employed with much care. There does not seem to be any one drug or any one means of treatment which is helpful in every given case.

There is one fact about the treatment of this disease which should be strongly emphasized; that is, the treatment during the acute period is *rest*—rest in as absolutely complete a form as it can be had. Also during the convalescent period the treatment is *rest*. The reason is plain: if the statement of the mechanical condition here presented as contained in the pathology holds good then anything that increases the blood pressure is likely to disseminate the infection again. The brain very likely acquires its immunity slowly. Finally, after the convalescence and for a long time, perhaps for a period of a year, rest is indicated again; so that rest, thus triply prescribed, is the surest and safest treatment we have knowledge of at present in dealing with this serious infection of the nervous system.

OFFICE ORTHOPEDICS*

WALTER TRUSLOW, M.D.

A GENTLEMAN was in my office not very long ago and said to me, "Doctor, you are an orthopedic surgeon; what is the meaning of that word 'orthopedic?'" I told him that it meant the care of deformities, the care of the crippled. I summed up by saying: "Anything crooked comes within our field." He said: "I wish you would come over to Wall Street."

The word is not from the Latin, *pes* (*pedis*), a foot, but from the Greek, *orthos*, straight, and *paedos*, child—to make the child straight. Orthopedic work was originally among children, but is now also for adults. There is, however, so much misinformation about the use of the word, that some of the orthopedic surgeons of the country have insisted that their work is surgery of bones and joints; and that is the field.

Orthopedic surgery has grown tremendously. We are almost in a position to say that permanent crippling is not necessary. Certainly many deformities formerly thought permanent are today cured. If you come to think of it, you do not see on the streets so frequently as formerly, persons going around with club-feet, for instance. The great epidemic of infantile paralysis of 1916 has somewhat modified that view, because it added a great deal more crippling. But much of that is being cleared up.

The general practitioner of today needs three things: more careful examinations, more careful diagnosis, and simpler methods of procedure.

I think it was Aesculapius, long before the Christian era, who was accustomed to sit in his beautiful gardens and have his patients come before him entirely nude. He would watch them walk about and get a great deal of information.

* This lecture was delivered on Nov. 10, 1922. When it was given, demonstration was made on living patients. The lecture seems best preserved in this series by adhering to its original form, although illustrations of each case may not appear.

That leads me to the first point, namely, the necessity, in office examinations, of getting the child, the man, and to whatever extent it is possible, the woman, with clothing off, so that one can observe them in walking, sitting, standing and lying down, and observe the motions and the function, as well as the physical defects.

How about the question of examining the woman in the office? There are a number of things that one can have that will be of real service to aid in such an examination. For instance, the office sheet: we are familiar with this. A middy blouse opened at the back can be worn, protecting the front and yet allowing much exposure of the trunk. The use of the towel, made into a kind of simple swimming tights, through the groin and tied with safety pins holding each side, makes it possible to do a good deal of examining in the office, and yet not let the woman feel she is unduly exposed.

Now, in examining, what do we see? We will have this little girl walk before us. We should look at the back to see whether, in standing, the shoulders are level. We should look at the hips to see if there is any difference in the contour, and we do find that in this case. We should watch the gait in walking. We are able from this to observe a number of things. Old Dr. Phelps, some thirty or more years ago, had a famous clinic which he called his "limp" clinic. He was quite skilful in imitating a good many of the different limps. Today the moving picture also helps us in this.

Now, we will let this child stand upon this bench. A bench is exceedingly valuable, especially when dealing with children. It brings the child nearer the level of the examiner's eye. Here, again, we may observe the abnormality of the two sides of the pelvis. Do we find on both sides what some one has called the "orthopedic groove?" That has been described as the depression between the crest of the ilium and the trochanter. It is distintly well marked here on the right side of this child. On the left side, however, there is a bulging—an obliteration of the orthopedic groove. If you look at the rear you will see there is a definite downward tilt of the hip on the left side. Something is there that is abnormal. As we find there is no pain, we shall probably be able to eliminate hip-joint diseases.

We have seen her walk; we have examined her standing. We will now examine her lying on the table. We will move the thighs up and down. There is no limitation on the right side. There is a little on the left. We obtain no pain in this passive motion.

We will see what the tape measure will reveal. You all know the points for measuring—from the anterior-superior spine of the ilium to the internal malleolus. The expressions R. A. and L. A. indicate these measurements.

I find here in this particular case 21¾ in. on the right: I find on the left 21¼ in. There is a one-half inch shortening on the left side, the same side in which you saw the obliteration of the orthopedic groove. We will turn the child on the side and trace Nélaton's line, which is drawn from the anterior spine to the tuberosity of the ischium. The trochanter is found distinctly above.

We have here a case of congenital dislocation of the hip.

Of course, it is wise to get an *x*-ray to confirm the diagnosis and to note the shape and size of the acetabulum and the head of the femur.

This is not a case for office treatment.

The method of reduction is a very definite one. It requires an anesthetic and considerable skill. But I do wish to emphasize the necessity of making an early diagnosis of congenital dislocation of the hip. It should be treated before five years of age, but if made a year or two later, it is sometimes successful. Let the parent's statement that the child limps warn you to make a thorough examination.

So, then, in our examinations of children we get much from watching their walking. We should observe limps, shortening and curves in the spine.

In the standing position we also can discover stiffness. Ask the child to pick up something from the floor. You will observe that this child stoops naturally and quickly and easily rises to the erect position again. If she should bend her knees with an erect, stiff back and should rise again with hand helping by resting on the knees, you would have reason to suspect disease in the spine.

In the borderline cases, where the stiffness and pain are not marked, I find it valuable to place the child seated in

front of me on the bench, allowing him to bend gently forward, and raise his trunk, my thumbs feeling successive vertebral levels. A location of initial disease can often be distinctly felt in this way. But it is safer to hold the child's trunk with your other arm, thus partly supporting the weight while he bends.

We will now put the child prone upon the table. On grasping the heels and extending the spine, we find in this normal child a definite, even backward curve, more marked in the lumbar region, but observable in the dorsal as well. The child, lifting its head, so as to look toward the ceiling, shows a similar free motion in the high dorsal and in the cervical spine. Lateral flexion of the spine in both directions also shows no resistance to motion. This is a child with a normal spine.

Always with a tubercular spine, or any kind of disease in the spine, there would be a rigidity showing itself more or less markedly and at the place involved.

Another valuable position for examination is what I would call half-prone lying at the end of the table. The trunk, head and arms are resting prone upon the table, the legs are perpendicular and beyond the end, and resting on the floor.

This is a valuable position for examining especially the low back and the sacroiliac joints. It is more used with the adult, where the trousers or the skirt somewhat loosened may still afford much protection to the individual, but leaving the back entirely exposed. In the case of sacroiliac strain, one may see a distinct bulging, may find and definitely locate a tender point, and may feel a bogginess at the same place. I know of no other position that brings that out so well.

While speaking of sacroiliac strain, and with this child as a model, I will mention a sign which I have found valuable. The man or woman complaining of low backache, is placed, standing, upon the bench. With direct listening with the ear or with the use of the stethoscope, placed on first one and then the other sacroiliac joint, he is asked to lift the knee and thigh sharply up toward the chest, and then more slowly to replace them. In true sacroiliac slipping, when the

thigh is brought back toward and nearly reaches the normal standing position, a distinct click is heard.

I would explain that as follows: If there is motion in the ilium on the sacrum, one gets a rotation of the ilium in this upward bend. It is actively controlled by the muscular action. But on the return, the muscles partly give up to the force of gravity, and at a certain place the ilium more suddenly slips into place again on the sacrum, giving the click that is heard.

In the examinations we had to make of candidates for the army, the Medical Advisory Boards had these cases brought to us to find out if a man was malingering or was a true sacroiliac sufferer. If he could not control himself at all, it was to my mind, a true test. The actual presence of that click in connection with the statement of pain and in connection with Goldthwait's sign, was to me one that absolutely indicated a case of true sacroiliac slipping or strain.

Dr. Goldthwait, of Boston, suggested some years ago, that the thigh be flexed on the hip and the leg on the thigh. There is no increase of pain in the low back because the hamstring muscles remain relaxed. If, however, we flex the thigh, but keep the knee extended, a sudden, sharp increase in pain in the region of the sacroiliac is experienced in the one who has such a strained or slipping joint. It is due to the fact that with the knee held straight, the hamstring muscles pull, from insertion to origin, upon the ilium and cause the ilium to move at the painful joint—sacroiliac. The three signs, the tenderness and bulging and bogginess felt over the sacroiliac joint, the direct auscultation in the knee-bending in the standing position with the Goldthwait sign of knee—straight, hip-flexing in the lying position, will almost surely make the diagnosis of this often difficult condition.

While considering methods of examination, we again refer to the valuable information we can get from the tape measure. In infantile paralysis, for instance, we should measure lengths of limbs, girths of thighs and of calves. Successive measurements taken at regular intervals record the progress of the disease. With persistent, and especially with increasing atrophy, we should feel that the methods of treatment used are not advantageous. With lessening atrophy, or, better

stated, with less discrepancy between the sound and the weak side, we may feel that we are giving the right kind of care—because nature does tend in this condition toward recovery, although improper treatment retards recovery.

We measure the length of the limbs; we measure the girth of the thighs and the girth of the calves. In measuring the girth of the thighs care must be taken first to measure down from the anterior-superior spine the same distance on both thighs; and this distance should be recorded that a similar measurement taken some weeks later may represent the same part of the thigh.

Again I would make a plea for more thorough examination of the patients that come to our office. Removal of much of the clothing will help us to discover what otherwise we could not.

Now, let us consider some of the simpler forms of treatment which can be given in our offices.

To return to sacroiliac strain or sacroiliac slipping—what is it? A true strain, usually due to injury from external violence, or to sudden violence in the individual's body movements. It consists of a stretching of ligaments and muscles, and sometimes of a tearing away of the sacroiliac cartilage from one or other of its bone attachments, leaving the ilium free to slip or slide a short distance away from the sacrum. It is the condition often called in the past "railroad spine," or just backache. It is not the only cause of backache, but is a great deal more frequently a definite lesion than has formerly been supposed. What shall we do for it? Hold the bones together until nature brings back the tone of the supporting structures. How shall we do this? The simplest way to begin, and to bring a large degree of immediate relief, is by pelvic strapping and sacral padding. Cut out a piece of felting, about the size of the sacrum. Place it over the sacrum and use many pieces of 2-in. adhesive plaster as follows: The first strip of adhesive plaster goes from, say, the right anterior-superior spine around the right side of the pelvis across the sacral pad, and then with a strong pull is carried around the left side of the pelvis to the left anterior-superior spine. The next adhesive reverses this. The next adhesive plaster parallels the first, overlapping a little

below it. The fourth adhesive plaster parallels the second, a little below it; and so one continues downward until the trochanters on both sides are included. The mistakes in adhesive pelvic strapping are that it is not carried low enough and the straps are not pulled tight enough. The sense of relief is often almost immediate. Because strapping stretches or slips along the skin, it is best to change about once a week. The average skin will stand this treatment for about a month. As the fixation of the joint must last many months, we must plan a carry-on which will avoid irritation to the skin. The special pelvic belt, with extra reinforcing—leather or webbing straps and buckles—is made and worn night and day. Your day laborer sometimes finds this out for himself. He removes his trousers belt and places it two inches lower at the orthopedic groove and finds he gets the relief. Very occasionally a plaster-of-Paris jacket is the only thing that will hold these parts properly together. Perhaps one in a hundred cases is so severe, or the ligaments are so relaxed, that for one reason or another, it is better to hold these bones together by inlay bone-graft operation. Sacroiliac strain is an exceedingly trying condition to the patient, but you will be much rewarded by his gratitude in giving him the relief from pain usually afforded by the pelvic strapping.

Next, we will discuss the after-care of infantile paralysis in relation to office routine.

We will not get the proper conception of the after-care treatment of infantile paralysis if we do not rate as of first importance protection of the parts involved—protection from strain and from deformity. At proper stages it is wise to give a mild degree of muscle stimulation by massage, by muscle training, and possibly by electricity; but the one treatment all the time is protection to the muscle that should not be made to function beyond its ability, and protection from the development of deformity.

This gentleman has been kind enough to let me illustrate an exceedingly common result of lack of protection—a result both in preventing the recovery of the muscles involved and in allowing a disabling deformity—club-foot—to develop. He had infantile paralysis in early infancy. He has had treatment in right lines spasmodically, but with

long intervals of poor treatment or none at all. Protection played but a small part in his previous care. He has consented to be operated on, and I will soon plan an operation which should practically overcome the deformity and allow a stable, even walking. Meanwhile I can show you a very valuable office use of adhesive plaster, as one of the simplest forms of protection of paralyzed muscles. This man has practically no power in the peronei of his left foot. His calf muscles have recovered, and his tibials have recovered. This unbalance of muscles inevitably causes the deformity here seen. The strapping which I will show you will allow this man to walk with much greater comfort and to wear his shoe more easily, although it alone would never overcome the deformity, and must be constantly renewed. The first adhesive plaster is placed on the inner side of the foot, short of the internal malleolus, under the sole of the foot, and with strong traction up the outer side of the foot and outer side of the leg. It begins the correction of the adduction of the foot at the ankle-joint. The second adhesive strip starts a little in front of the first, overlaps it and extends parallel with it. It continues the partial correction of the adduction of the foot and begins the partial correction of the varus of the forefoot. Each succeeding strapping starts a little further forward and extends upward on the outer side of the foot; and each strapping is further corrective of the varus of the fore part of the foot. Any strapping of a foot must avoid complete circumference so as not to limit circulation. This strapping might be considered as a definite reinforcement of the weak peronei muscles. It points out the principle of all such strapping for paralytic conditions. The strappings of adhesive plaster are placed over the muscles that are weak, and the part acted upon by such muscles is held in the position which most relaxes the weak muscle. Plaster-of-Paris dressings or braces would supplement and replace strapping for longer use. Personally, I am more apt to use bracing in the summer time, when daily removal for washing is more indicated; and plaster-of-Paris in the cold months of the year, when the warmth of the under-padding is greatly indicated to combat the tendency to coldness of the part.

What about flatfoot? An orthopedic surgeon is not a "foot doctor," but he has a great deal of foot work to do. You all see these painful feet. What do you do with them? This boy whom I show you will make it possible for me to demonstrate the simplest procedure—that of padding and strapping. He is placed sitting on the table with the thigh and leg "side-saddled," the foot protruding just over the edge of the table. First, we prepare the felting pad. It is not to support the plantar arch alone; it is not to support the metatarsal arch alone. It is to be cut in an irregular shape to fit the normal hollow places between the three supporting points of the foot—the heel, the ball of the great toe and the ball of the fifth toe. The two arches of the foot, the plantar and the metatarsal, are merely two parts of the continuous hollowness, which should be filled in its entirety by the pad, cut as I show you here. The foot-braces found at shoe stores may give a fair support to that portion of this hollow which we call the plantar arch. One begins to see today another form of store foot-brace which presents a lump under the middle of the ball of the foot and is supposed to support the metatarsal arch. Neither one of them is a sufficient support of the foot. They are often exceedingly harmful.

This felt padding and, incidentally, any form of foot brace that is used, should combine support to fill up the entire hollow which has been discussed.

The felting, prepared, is placed in its proper position on the sole of the foot and the first adhesive strapping—inch-wide adhesive is the best size—is applied. This first adhesive extends from the outer side of the foot, short of the external malleolus, underneath the sole of the foot, partly fixing the felt pad, up the inner side of the foot across the internal malleolus, and about an inch up the inner side of the leg. The long straps far up the leg are seldom necessary. This first strap begins the correction of the abduction at the ankle. The second strap is transverse to the first and goes from the outer side of the heel, across the back of the heel, across the inner side of the foot to the dorsum of the foot. It should be placed with the heel and the forefoot valgus as much corrected as possible. These two strappings repre-

sent the correction of the deformity. Actually, they are not strong enough, and are reinforced with many parallel strappings placed criss-cross to each other and further correcting both of the features of the deformity indicated. Practically, it is wise to strap and pad the average painful foot for about a month, changing the strapping and padding about once a week. By the time the last strap is removed the foot-brace, specially made for the individual, should be ready.

The details of foot bracing seem to me beyond the scope of this lecture, but the principle of foot bracing should be this: A foot should be braced until it is strong enough to bear the weight of the body without pain. This is accomplished only by the third procedure in the care of flatfoot, namely, the daily ten-minute-a-day intensive foot exercise. Printed cards of these foot exercises such as I use are of exceeding value. They include:

SITTING WITH FEET APART AND PARALLEL

1. Raise the inner borders of the feet without moving the knees..... 10 times.
2. Raise the fore part of the feet; turn the toes under and pivot (on the heels) until toes touch... 10 times.
3. Place the right foot over the left knee and turn the sole upward..... 10 times.
4. The same with the left foot over the right knee 10 times.

Nos. 3 and 4 are often aided by forcible stretching with the hand.

One hour is little enough time to cover even that part of orthopedic work which is cared for in one's office. If, however, I have been able to show methods of examination and emphasize the value of thoroughness in examination, and if I have demonstrated a few of the simpler procedures which are most common in our practice today and which any one with patience and a little mechanical skill can master, I shall be content.

MEDICAL PSYCHOLOGY*

JAMES J. WALSH, M.D.

I NEED scarcely say that I am not going to try to exhaust the subject chosen for this lecture. I have written a volume of half a million words on Psychotherapy, which is just one phase of medical psychology; so that, at least, I know the extent of the subject. Nothing is so likely to spare an audience a long-winded dissertation on a theme as to have the speaker know something about it. A man who had spent many years in lecturing declared that the all-important qualities in a lecturer were to have something to say, to say it and then stop. The trouble is that most of us are not very clear about what we have to say, and we scarcely know when to stop. A western railroad man, who attended some of our New York banquets and listened to after-dinner speakers, said that they seemed to forget that the important thing for a railroad and for an after-dinner speaker is terminal facilities, and that most men seemed to have very inadequate terminal facilities.

I shall never forget an old professor who had taught me chemistry but was not sure that I knew very much about it, asking me as a leading question to tell him about oxygen. I was not sure where to begin, so I said, "Do you want me to tell you all about oxygen?" He promptly replied, "Oh no, only what you know about oxygen." I am not even going to tell you all that I know, or think I know, about medical psychology. I am going to try to give you a simple introduction to a very large subject. I am quite sure that a great many people here know just as much about it as I do, though some of them may not have been forced to organize their knowledge and make it available for suggestion to others, to the same extent as I have done. That is the only reason why I am here, to try to tell you some things that you yourselves do know, to use Antony's expression, and

* This lecture was delivered on May 11, 1923.

ask you to apply these ideas in such a way as to bring about the healing of disease, which is our very convenient word for the discomforts of mankind consequent upon particular affections. We talk about men and women suffering from complaints, and that is another strikingly convenient word. There is no question of curing organic disease, and yet of doing a great deal of good in the saving of suffering and even in the prevention of such disturbance of function as might produce serious effects.

Probably the most interesting phase of the history of medicine, certainly the most human and humorous, is the chapter that might be entitled, "The Cures That Have Failed." Physicians at least will agree with me, but so will all those who know even a few of the facts. All down the centuries we have had remedies of one kind or another, modes of treatment of all kinds, physical, chemical, herbal and mineral, applications of various kinds, treatments direct and *in distans* which have "cured" people, according to what is presumably the best possible evidence—their own declarations—of all sorts of ills that flesh is heir to. The strange thing is that after a comparatively short time these vaunted cures have proved to be utterly inefficacious, and indeed have not infrequently been demonstrated to be positively harmful rather than beneficial in their effects. Here is one of the supreme paradoxes of history from one viewpoint; and yet it is only a commonplace of human nature to the student of psychology and of psychic processes. All this is so familiar to physicians that one almost hesitates to talk about it, and yet it seems necessary to do so; for not only our patients but also we physicians ourselves are taken in by these cures, which indeed are so insidiously presented as to deceive even the elect. The woods are full of the cures that have failed, not only in the hands of the quack and the charlatan, but also of the scientifically trained physician.

A great French therapeutist once said, "The cures of any generation are always absurd to the second succeeding generation." In recent years, when the news of remedies spreads more rapidly than in the past, and when carefully controlled tests of them are made within a short time of the original announcement, most of these tests being conducted

quite apart from the atmosphere of favorable suggestion that influenced the cure at its birthplace, it does not take so long as the second succeeding generation for us to find out the truth. As a result, we often discover that remedial measures that have been praised to the skies as eminently curative, prove, on further investigation, to have no salutary effect at all, or perhaps but the very slightest. Yet when first employed, especially by their original discoverer, they seemed almost to work wonders of healing and to produce results that had been devoutly wished for, but had not been secured before. It is just a question of the over-enthusiasm of the inventor and the consequent prejudgment of the significance of events, that makes him see only the favorable evidence while neglecting, though often quite unintentionally, the unfavorable. Besides, to one who is intent on finding something, who has the will to believe, in Shakespeare's words, "trifles light as air become proofs as strong as gospel truth."

A typical example of the influence of the "will to believe" on physicians and patients is to be found in a story that was popular in Paris when I was making my medical studies there something more than a quarter of a century ago. The story seemed to the French to have all the more point because of the then recent failure of Koch's tuberculin. The greatest of living bacteriologists after their own Pasteur had announced at the International Medical Congress, held at Berlin in 1891, a cure for tuberculosis which, for a while, had seemed to work the wonders that was promised for it. However, on further investigation it had proved to be not only an utter failure as a cure, but a rather dangerous remedy for many tuberculous people to take. It exhibited, in many cases, a definite tendency to make the tuberculous process acute and to produce a wider dissemination of the tubercle bacilli, sometimes with the actual production of *phthisis florida.*

With that in mind the biting Gallic wit of the story will be more readily appreciated. A young physician, suffering from tuberculosis himself, consulted an old colleague who had been his professor at the University of Paris Medical School, for advice with regard to his condition. What he wanted to know particularly was whether he should take a certain new

remedy which had recently been announced as a "sure cure" for tuberculosis. The old professor, out of the wisdom of his years of disillusionment with cures in the practice of medicine, said to him, "Oh, yes, take it by all means, and take it now while it still cures, for after a while it will be found not to cure, and then, of course, it will do you no good."

We have had any number of remedies and modes of treatment all down the centuries, of which this same thing might have been said very aptly. They have cured people of all sorts of ills and have relieved patients of most of the affections to which human flesh is heir, and a few others besides, and yet have since found their way into the discard. There is sometimes the feeling in our generation that this was true only in the older time when we knew much less about scientific medicine than we do at present, and when physicians and patients were necessarily dependent on remedies that were at most empiric, and before such development of the science of therapeutics as enabled us to measure and estimate exactly the value of remedial measures. Any such thought as that, however, may be dismissed at once; for there was probably never a time in the history of medicine when so many people were cured of ills of various kinds by cures that afterward failed or that manifestly have in them no physical effect or only the most trivial efficaciousness as medicines. Education is supposed to have made people proof against delusions of various kinds, and especially against self-deception as regards disease; but as a matter of fact it has not. Professor Dewey said, not long ago, that the best criterion of education we have is that it keeps people from being duped, but that, tried by this criterion, education in our time was not only bad but is growing worse. "For this is the age of bunk and hokum," and this expression of his is true particularly with regard to medicine.

Macaulay, now nearly a hundred years ago, bewailed the fact that a great many of the educated people of the London of his day, some of them members of the nobility, not a few of them graduates of colleges and universities, became disciples of Johanna Southcote, a scullery maid, who after having had a series of revelations as the result of which she cured the chronic ills of those who believed in her, at the age

of more than sixty announced that she was to be the mother of Shiloh, "the man child who was to rule all the nations." Her pseudocyesis delusion was undoubtedly due to the fact that she was suffering from Bright's disease and that dropsy was developing. Shiloh failed to appear, of course, and it was announced that Johanna was in a trance in communication with the Most High; but before coming out of it she died of coma consequent upon her nephritis. Even these events did not undeceive her followers. She had worked "miracles of healing" and, therefore, the people were quite sure that she must have some very intimate association with the other world. Macaulay said: "We have seen an old woman with no talents beyond the cunning of a fortune teller and with the education of a scullion, exalted into a prophetess and surrounded by tens of thousands of devoted followers, many of whom were in station and knowledge immeasurably her superiors; and all this in the nineteeth century; and all this in London." Macaulay resented it particularly because it took place in England, above all in that very heart of civilization, London. Had it occurred anywhere else he would probably have felt that no more could be expected of other peoples, but that the cultured English, even the nobility, should fall this way seemed to him inexplicable. Some of you will doubtless recall that the dear old nineteenth century thought as much of itself as we of the twentieth century do of ourselves, but then that has always been so. Each ultimate generation is more than a little contemptuous of its predecessors and self-complacent about itself.

Had Macaulay been alive some hundred years later, that is to say, in our time, he would have found, in spite of the development of education, of which we are very properly so proud, just as great a tendency to follow after curious healing cults as shocked him in the case of Johanna Southcote in his day. At the beginning of the twentieth century, Dowie, who claimed to be Elijah returned to earth to prepare the way for the coming of the Saviour, cured his tens of thousands. Most of them had money at least, and the making of money is supposed to be an index of intelligence, though Mark Twain suggested that "dollars and sense" by no means necessarily go together. Macaulay could have

written another chapter in the twentieth century on Francis Schlatter, who died only last year, and who pretended to have fasted for forty days, thus acquiring wonderful closeness to another world that enabled him to heal people by his touch. Indeed, for a time he had a very lucrative correspondence course, in which he mailed handkerchiefs blessed by his hand to those who could not come to see him. The touch of these cured many people, according to that best of all evidence, their own declaration. It is to be supposed that these were used handkerchiefs, surely containing emanations from the healer. Unfortunately for him, the United States Government declared that handkerchiefs at $10.00 *per* were an imposition, so the postal authorities issued a fraud order preventing the further development of this very interesting therapeutic departure.

It would be amusing to know what Macaulay would think of the so-called religious meetings at our most important hotels in New York City every Sunday. The Saturday papers make announcements of a wonderful variety of religious services. Some of them are legitimate church advertisements to catch the eye of our large floating population of visitors, some half a million in number, at times; but most of them are just "healing religions," meant to catch the susceptible and suggestible. The healers talk in the ballrooms or in the larger parlors of the big hotels. Needless to say, only those who are well dressed, who show external evidence of good breeding and who are presumably intelligent are welcome. These "religious" leaders heal by all manner of means. "You pays your money and you takes your choice." They have various forms of metaphysical healing and psychological healing and healing through the influence of spirits and healing through many kinds of oriental cults. Talk about "varieties of religious experience"; it is perfectly possible to get nearly all you want of them without leaving the beaten track of Broadway in the neighborhood of the great stations. They call them religions, but some of them have very curious names and still more curious and mystical connotations. There is, for instance, the religion of the solar plexus and the religion of the ductless glands, as well as healing through the mystic influence of the sym-

pathetic nervous system, and so on. It is quite evident that many people are healed; for the healers make a good living. They often reside at the hotels where they hold forth; and that cannot be done for nothing, besides paying for the meeting rooms.

Manifestly it is not a question of intelligence, but of certain natural tendencies in mankind. We are all prone to take suggestions, and when we are ill we become more suggestible. This tendency produces certain symptoms in us and these symptoms can only be cured by suggestion. The war proved very illuminating in this regard. There was a general impression that hysteria was a disease that affected women almost exclusively. Physicians knew that occasionally men, presumably with more of their mother in them than they ought to have, suffered from hysterical symptoms. Such cases were supposed to be quite rare; and one scientific explanation of them was that by a confusion of morphological elements in genetic development certain males had unfortunately been accorded an admixture of female elements, which gave them feminine tendencies. Since every human being is a result of a conjunction of the sexes it is possible that there might be an error of this kind—the delicate balance of embryonic life might be disturbed by some accident—but this was an extremely rare event and not to be considered in the ordinary outlook on disease.

During the war, however, we had in this country and among our young soldiers in France many thousands of cases of "shell shock" which were almost without exception pure hysteria. It is true that a few of them were possibly real concussion of the brain due to the explosion very near by of a high explosive shell. A certain number of others of the "shell shock" cases were definite mental diseases which had been brought on by the stress of war before the time when they would ordinarily have developed, in persons with unfortunate heredity. The great majority of them, however, were functional neuroses, usually of the type known as psychoneuroses. Here in America we had realized from the experience of the Allied armies, as well as those of the Central Powers, that such cases would be very frequent. We were inclined to think, however, that we had rather

the advantage of the Europeans in a certain stability of nervous system, as the result of which nervous systems in this country were likely to be able to stand up more thoroughly under the stress and strain of war. We were shocked to find, however, that this was not so. If anybody had told us before the war that the most important official in the army medical service, after the surgeon, would be the neurologist, we would not have credited it. We had "shell shock" cases by the thousands, however, and the Allies had to provide literally tens of thousands of beds for these hysterical young soldiers behind the lines in France. There will be plenty of room for a variant on Bernard Shaw's "Chocolate Soldier" when we can look back impersonally upon our war experiences in this regard.

The British experience was like our own: they found that there were nearly four times as many officers who suffered from "shell shock" as privates; which means that education, instead of making one less susceptible, actually makes one more prone to suffer from this psychoneurotic condition.

The story of M. Coué is very interesting as a sidelight on our civilization and progress. At the beginning of the second decade of the twentieth century this very complacent little druggist comes over to this country and receives as much attention in the newspapers as any of the most distinguished visitors from France since the war. He gave a series of lectures which crowded the largest halls to the doors, breaking the records for attendance at most of them. He proclaims that he is not a wonderworker and that he does not cure people, but that he teaches them how to cure themselves. Anyone who went to one of his public lectures will know that the audience, in spite of this very candid declaration, waited almost breathless with attention waiting to see him do marvels. Whenever they could get near him they had a certain satisfaction in getting into his presence, as if his very passing near them might do them good. They had the will-to-believe and the will-to-be-cured quite as strongly developed as ever had any medieval audience. Manifestly, a question of human nature was involved, and this is just the same in our time as at any time in the world's history, unmodified by education, civilization, the progress of the race, or any

other of the achievements that are supposed to have come to us.

M. Coué is a very interesting man to study, because he has reduced this whole question of healing to its lowest terms. Healers of all kinds have worked "cures," but, as a rule, they asked that you should accept something that was quite absurd on the face of it. Dowie and Schlatter in our time, as commissioners from on high, or Greatrakes in Harvey's century, as divinely commissioned by the Holy Ghost, required an act of faith in their divine mission. When Perkins cured people with tractors that had no electricity or any other force in them, he demanded an act of faith in the marvelous electrical power of metals in contact. When hypnotism made its cures, all the world was persuaded that hypnotism was a wonderful process that enabled a strong-willed individual to give something of his energy to others less well endowed than himself. Now that we know that hypnotism is only induced hysteria, it no longer makes cures. Always, up to this time, for a cure to be effective there had to be an act of faith in absurdities. This continues to be true. A great many people are cured by Christian Science, but only by believing that disease is an error of mortal mind and has no real existence. Indeed you are supposed to accept that matter has no existence and that you cannot have anything the matter with you, because there is no matter in which to have anything the matter with you, and that all you have to do to be well is to stop believing wrongly that you are ill, and then all will be well with you.

Many people are cured by osteopathy, but they have to accept the idea that practically all the ills that flesh is heir to, more than 95 per cent of all our diseases (as Dr. Still said when I had a little controversy with him some twenty years ago in The Independent), including typhoid fever, cholera, dysentery (bloody flux as he preferred to call it) cancer, tuberculosis and all the rest, are due to a subluxation of the vertebrae that brings about interference with the blood and nerve supply to the parts that are affected. The act of faith is just as necessary as it was three hundred years ago, when the weapon ointment was making its wonderful cures, that, too, had to be believed in. This ointment was guaranteed to cure

any wound provided it was applied in time, but it must be applied, not to the wound, but to the weapon that made the wound. Undoubtedly it did a great deal of good in a negative way, because wounds had been treated by messy salves and ointments of various kinds. This, at least, prevented that abuse, which had inevitably infected many wounds and made their course of healing much longer and more severe than they would have been without them. The ointment did so much good that even that great founder in physical science, Francis Bacon (wrongly called Lord Bacon), was inclined to believe in it. Such an act of faith could be curative.

The most surprising thing about these cures—of which the history of medicine is full from the very earliest traces of interest in bodily affections down to this year of grace 1923—is that the affections cured by the healers are mainly those that would seem to require very definite physical means acting directly upon tissues to afford relief. For it is particularly the pains and aches of mankind and the disabilities of many sorts, that have been cured by all kinds of means that afterward proved entirely inefficacious. The group of affections that used to be called rheumatisms, the pains and aches that are worse in rainy weather but that have none of the other classic symptoms of arthritic or muscular rheumatism, are above all those that were cured in this very interesting way. They had the *dolor*, but not the *calor*, *tumor* nor *rubor* of Galen's well-known category of symptoms; but the patients, as a rule, complained enough to make up for the other symptoms, and often they could not use their muscles without an amount of discomfort quite disabling.

These are the patients, above all, that have, down the centuries, given reputation to all manner of remedies and healing modes, and conferred prestige on all sorts of healers. When hypnotism, at the end of the nineteenth century, came in as a therapeutic resource at Nancy, it was because in Liebeault's hands it had been curing the lumbar and upper-leg pains and aches, the lumbagos and sciaticas and the shoulder girdle pains that so often prove crippling for men and women. These were the patients cured by blue glass in the second half of the nineteenth century, and by magnets and little Leyden jars in the second half of the eighteenth

century. It was cases of this kind that were cured by Perkins with his tractors in Connecticut, and by his son in England. Of course it was this sort of patients that gave a reputation to St. John Long's liniment and to Mesmer's healing efforts, though it was also such patients that formed a large part of the clientele of Dowie and Schlatter in the twentieth century. I need scarcely tell you that it is just this same class of patients that now flock to M. Coué and are cured by him. I believe that many thousands go every year to his clinic at Nancy and that 60 per cent of all that go are cured and 90 per cent are relieved. The more you know about his cases, and especially his success, the more you realize that it is of discomforts of various kinds that his patients are cured. Many of his cures indeed are of the curious muscle pains and the disabilities associated with them which constitute the material out of which healers have made their reputations all down the centuries.

Our conclusion is that the discomforts of humanity, the complaints of the ailing, must, apart from definite evidence to the contrary, always be looked upon as subjective rather than objective. A comparatively slight discomfort, when a great deal of attention is paid to it, may very well become an almost unbearable torment. There are two reasons for that. One is what Ramon y Cajal, the great Spanish brain anatomist, called the law of avalanche. The other is the reflex action of attention upon the tissues to make them more sensitive than they were before. What Ramon y Cajal meant by the law of avalanche is best illustrated by an example: If we have a painful condition in a finger, a certain number of cells in the brain in connection with the part are rendered uncomfortable; or, if that does not sound psychological, produce a sensation in our consciousness of distinct but limited discomfort. Unless it is very severe we can forget about it by occupying ourselves with something interesting, and thus be absolutely unaware of its presence for hours at a time. If we have nothing else to occupy ourselves with, and if we turn all our attention on the painful part, fearing that its consequences may be serious, or if, for instance, we have been bitten by a snake and we are afraid that the poison of it may be spreading through our system with possibly fatal result,

our attention is actually concentrated on the affected part; and, while before, a few hundred or only a few thousand cells were carrying the complaint to our consciousness, we may now have hundreds of thousands or even millions of cells preoccupied with it. If we are intensely afraid of the consequences, every possible cell of our brain may become connected with the area directly affected by the discomfort, and there may even be billions of cells disturbed by it.

Hence the figure of the avalanche suggested by the Spanish anatomist. A few stones or pieces of ice start rolling down the side of a mountain, they loosen some others, and these in turn loosen others, until a hundred or more are finding their way down the mountain side. These in turn, loosening others, finally gather a great mass of materials that can prove destructive to life and habitations and even villages or towns that may be in the way. A very slight cause may produce an immense effect. A comparatively slight discomfort, if there is no diversion of mind, but instead a concentration of attention (as often in suggestible people, who, instead of hoping for the best are always dreading the worst), may cause the development of a condition in which patients are intensely miserable. Yet the physical element in their condition is but very slight, and only the neutralization of their state of mind will enable them to improve.

The other factor increasing discomfort is due to the phenomena by which, whenever attention is paid to a particular part of the body, it becomes much more sensitive, or at least responsive to sensations, than before. If, at a given moment, you think of the middle of your back, just below your belt, you will find sensations there that you had no consciousness of a moment ago; and if you continue to think about it those sensations grow more noticeable. Further dwelling on them may even cause the feeling that something must be done to relieve the tingling and pressure; and to keep from having to solace the feelings by itching or rubbing, or some other external mode, is not easy. On the other hand, if you think of the outer surface of the big toe on your left foot you will notice that your shoe and stocking are pressing upon it. These sensations will grow the more you think of it, though you will notice at the same time that the trouble in

the middle of your back has disappeared completely. This shows how much diversion of mind can accomplish at once. And now your toe may become so insistent in its claims upon attention that you will at least want to rub it against the other shoe in order to secure some relief from the feelings brought to your consciousness as the result of attention to it.

In a great many cases people have only some comparatively slight disturbance of sensation. Sometimes it is scarcely more than a physiological feeling inevitably consequent upon function, and yet by paying attention to it they are exaggerating their discomfort into a torment. The old German professor long ago suggested that people did too much coughing because every little sensation in their lungs during the course of a cold they took to be an index of the need for coughing. As coughing requires a convulsive effort, it disturbs the lungs, especially when they are not well; and while it is very valuable as a means of getting rid of material, it should not be used except when one is required to choose between two evils—the cough, or retaining material that ought to be gotten rid of. Then, of course, the lesser evil is chosen. It was because of this that the old German professor said that no one ought ever to cough, as a rule, or to scratch the head, unless these activities were productive. If you got something for them, then they were justified, but scarcely otherwise.

I know that to many people any such explanation of complaints will seem entirely too trivial to account for the symptoms which they think they have. Once more, I can only refer to the fact that the cures that have failed show us very clearly how many people must have been suffering from complaints of this kind, or they would not have announced themselves as cured by the very trivial means that were employed on them. Of course they thought that some new and wonderful discovery or some entirely novel method of treatment had been found, and that they were obtaining the benefit of it. This occupied their attention so much that it was comparatively easy for them to divert their minds from that concentration of consciousness on their discomfort which had characterized their mental state for a prolonged period. Just as soon as this diversion of mind came they

began to feel better. With the first sign of betterment they began to eat better and to get out more in the air because they felt that they were recovering, and then a whole series of habits that made a virtuous circle—air, exercise, food, occupation of mind, thinking that they were going to get better, feeling light-hearted because of it—were so set at work for their betterment, that it was not long before they felt much better. This encouraged them to look for further improvement, until in the course of a comparatively short time they found themselves entirely well.

Of course the root of most of these pains and aches and disabilities is a fear of something of serious nature developing. A man bumps his knee against a post or a door in the dark, and the direct injury itself proceeds to get well, but when it is practically all recovered it is noticed that he has begun to favor that leg. This may go on until he walks lame, and as the result of disuse, even a certain amount of atrophy takes place. This may be entirely a psychoneurosis, that is, a neurotic condition of the muscles around the knee consequent upon his dread of using it lest it should hurt him while the injury was yet producing some pain. Such a man will not regain the use of his limb unless some very strong impression is produced upon his mind. This may be done by a bone setter or by the touch of a magnet or some wonderful new liniment such as St. John Long used, or by electric applications in some form; but the change in his mental attitude must come before he will recover.

The same thing may happen with regard to a true sciatica or lumbago. The inflammation within the sheath of the nerve will cause the disability, and the use of other muscles will be accommodated to the limitation of the use of the muscles supplied by the affected nerves, and thus are acquired certain bad habits which may become inveterate. I have known a very distinguished mathematician, a man of the very highest mental caliber in this country, to wear crutches while recovering from a sciatica, and not be able to give them up, though some of the best physicians in the country declared that there was no reason in the world in the condition of his nerves or muscles why he should continue to carry his crutches. Lumbago often does the same thing—

leaves a crippling, due to the patient's state of mind as regards his muscles, and his dread that they will be painful if he uses them normally. This is why lumbago has been cured by magnets and by plasters of various kinds, which really were scarcely more than rubber plaster; or by wearing a sheet of paper over the back, as Liebeault used to insist with his patients after hypnotizing them, so as to continue the suggestion of cure by the continuous sensation of the paper.

The same series of events may follow a sprain or a dislocation where the part has been set completely at rest. There will be some atrophy of muscles from disuse and it is extremely difficult to win them back to their former state of nutrition. Young folks can do that very well, but older folks balk at it; even those of middle age and younger form habits of sparing the muscles or using them wrongly and inefficiently, because they are unable to push through the period of pain which will follow their first attempts to use them. Many such patients use crutches and canes and other aids to locomotion long after they need to do so; and the physician must actually often be quite stern and firm in order to get them back to the normal employment of their muscles.

What is true for the external or muscular functions of the body is even more true as regards the internal, and above all the digestive, functions. Some disturbance of these is almost inevitable every now and then, considering the possibilities of spoiling food, the incompatible mixtures and the idiosyncrasies. Having suffered from one of these, some patients become alarmed and proceed to think that a passing symptom is an index of a serious progressive disease, or at least some unfortunate development in tissues. The result is panic, then failure to eat properly. They gradually eliminate from their diet everything that they have ever heard of as being indigestible in any way, until they are likely to reduce seriously their nutrition. They eat less than they ought, and their stomachs, often craving more to do, and abhorring a vacuum, proceed to fill the hollow digestive organs with gaseous content that has leaked out of the blood vessels in the walls of the gastrointestinal tract. Then the patients suffer from the added symptom of gas, which they suppose

to be due to fermentation, and a vicious circle is formed. They cut further elements out of their diet and this leads to the development of constipation; then you have neurotic gastrointestinal indigestion with its complications and sequelae.

I do not know how many nervous patients I have cured by simply getting them back on their "feed." They are not eating enough. This gives them a sense of inadequacy as to their tired feelings, keeps them from sleeping at night and is the basis of gaseous distention and eructation. The whole symptomatic picture, which sometimes has lasted for years, will clear up completely under proper amounts of food taken three or four times a day at regular intervals. When people are thin, the adding of 10 per cent to their weight, with the feeling of pep that accompanies it, makes the best possible kind of a suggestion carrier. Every time they record another pound on the scale they know that they are getting better; and without having to be told to do it, they wake in the morning and say to themselves: "Every day, in every way, I am getting better and better." Before going to sleep at night they congratulate themselves on this fact; so, of course, they get better. It is surprising how many people are giving themselves unfortunate suggestions with regard to health, making themselves miserable and really producing certain bodily conditions, who get better at once when they are given a proper suggestion, if they only take it. For that, they must have confidence in their physician and a certain willingness to do what they are told in the matter of air, exercise and diversion of mind; but with these they proceed to get better.

FUNDAMENTAL SIGNS IN THE EXAMINATION OF PATIENTS*

LUTHER F. WARREN, M.D.

WHEN Dr. Gordon asked me, some time ago, to discuss this subject, I realized it was a difficult one to present. I would have preferred some such subject as the differentiation of fevers, or a series of headaches, discussing the etiology and findings, these being clinical subjects which one can follow out and correlate as he goes along. But the subject of physical diagnosis was rather stressed by Dr. Gordon in asking me to attempt it, and I accepted his wish as a command, because I felt that every one should do his part in helping to carry out this series of lectures.

I

We all know that physical diagnosis is a subject which is not taught by reading, nor by looking, but it must be taught by one who has received instruction in it in such a way that he can give it to another. It is a subject that is taught from eye to eye, from finger to finger, from ear to ear, or from nose to nose. For instance, a student is told that this is a gallop rhythm and he listens until he can say, "I hear as you do"; he then has the fixed impression of it and thereafter he can recognize it.

In considering this subject I must of necessity limit myself, for if I should start to describe signs, even taking up only one particular sign, I would fail to convey to you an impression exact enough to have you recognize it the next time you met it in practice.

This subject, then, is the art of our profession, and as an art it has no symbols definite enough to be transmitted in descriptive words. I do not plan to give you a compressed lecture on the interpretation of physical signs, but I do hope to give you some facts here and there, if you will allow me to

* This lecture was delivered on Dec. 8, 1922.

do it that way, on this very important subject. With this in mind, during the last six weeks, I have screened material which will help to bring out the points I am going to discuss.

The first point in the study of the individual carries me back a little from my subject, which is especially physical diagnosis. There are three parts of every problem case that comes to us: the first is the clinical history, the second is the physical examination. These two furnish the data upon which we should ordinarily be able to formulate a clinical opinion upon an individual case. The third part is the investigation of special fields; they may be laboratory fields or fields of the specialists; but the laboratory men and the specialists work to confirm our opinion or to negate it. Our part is to examine and to obtain findings which, correlated with the history, make it possible to give an opinion as to the patient's condition. Further than that we call upon special assistance to aid us. When a patient appears and says, for example, "Doctor, I have palpitation," if we resort at once to the stethoscope, we go quite astray, for the history should give us the guide marks, not only as to our findings and what we should really look for, but as to the interpretation of those findings. So, before starting with the physical examination, I propose to go back a little in order to emphasize the importance of the history.

If one studies for a moment the summary in Figure 111 of the clinical physical findings, it would be reasonable to state that the patient had a bronchopneumonia. She did have it. But if we stopped there we would misinterpret the meaning of the signs in this individual case.

Case 1. (T. 103, P. 120, R. 36) Female, married, aged thirty-eight.

History Summary. No severe childhood diseases. Married at twenty, mother of four children, the last born twelve days ago. No complications following previous births. Five days after last birth severe chill, lasting one-half hour. This recurred at irregular intervals, twice since. Five days ago cough appeared. Then began to expectorate yellowish sputum.

That history of her illness, beginning five days after delivery, made us investigate her pelvic findings more in

detail, and we found that she had a thrombophlebitis of the pelvic veins. We grew a hemolytic streptococcus from her uterine discharge and the same organism from the sputum. Thus we finally interpreted the physical signs, through the added points brought out in her history, as a bronchopneumonia, secondary to her pelvic infection. This infection had been carried through the blood stream and had been sieved out in the lung bed, giving rise to her pulmonary

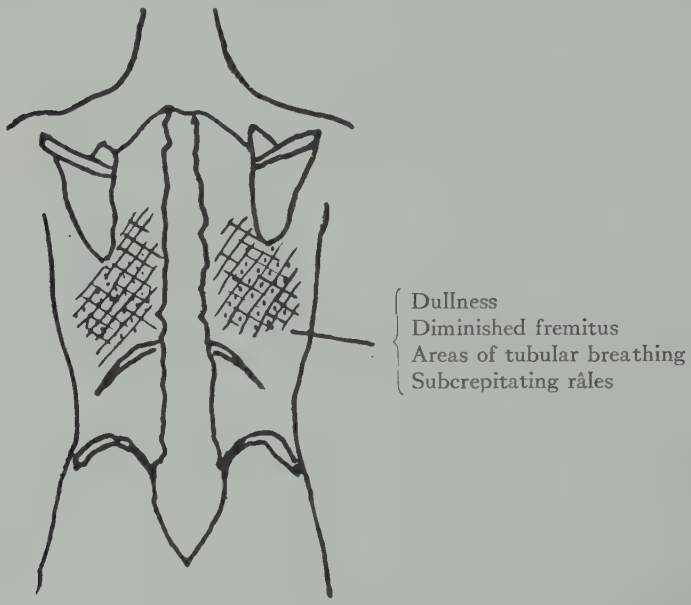


Fig. 111.

disease. The course of the case substantiated this interpretation. She ran a temperature for five weeks, and finally the lung findings, which were variable, but present during the entire time, cleared up and she recovered.

Figure 112 emphasizes the necessity of interpreting the physical signs through the clinical history.

The above physical signs are indicative of exudate in the bronchial tree, but are also quite classical of fluid in the pleural cavity. Naturally one's next inquiry would be: what is the nature of this fluid? Aspiration of the cavity was made

and pus obtained. Hence, although the man had empyema, he had much more than empyema. His real pathology and the cause of the empyema could be correctly interpreted only through the history which follows.

Case II. T. 104, P. 120, R. 25. Male aged thirty-nine.

History Summary. Pneumonia, 1918; malaria, 1920 and 1922; amebic dysentery, 1920 and June, 1922. Chancre two years ago; four injections of salvarsan.

Fig. 112.

Chief Complaint. Bloody sputum; general weakness; slight pain in right axilla—one month duration; thick pink-tinged pus aspirated from pleural cavity—sterile.

As we go over the history we find there are some points of importance in interpreting his physical signs. We find he had had amebic dysentery in 1920 and again in June, 1922. We then turn to see if there is any possible relation between that infection and his present condition, which began insidiously with cough and the spitting of blood-tinged sputum, general weakness, and slight pain in the axilla. We looked for amebae in the stool and sputum, but none was found. Now

the fact is that, although the man had sterile pus, he had a liver abscess. This had ruptured, thereby causing a subphrenic abscess, and finally had given rise to his empyema.

If one had not investigated or interpreted his physical findings through his history, one would certainly have missed part of the real issue. I am glad to say that this case was diagnosed correctly as shown by necropsy.

Case III. Figure 113, a female, aged fifty, is a third case to illustrate the point I am trying to make.

Physical Summary. Blueness of both feet. Blueness of right ankle extending up the inner side of right leg. Remain-

Miss G. Age 50. Blueness of both feet. Blueness of right ankle extending up the inner side of right leg. Remainder of either lower extremity white and cold to knee. Sensation lost completely. No pulsation in bloood vessels. No edema. No temperature.

FIG. 113.

der of either lower extremity white and cold to knee. Sensation lost completely. No pulsation in blood vessels. No edema. No temperature.

If we stopped at the above physical summary we could say very little. One might say she had a slight gangrene as the result of some arterial change, or possibly angiospasm, or even Raynaud's disease; each of these opinions had been offered. But if one investigated the history one found that she had, four days prior to entrance, a very severe pain in the right popliteal space. It came on very suddenly; it was the most severe pain she had ever experienced; it was like a knife sticking into her.

She could not walk, and was placed in bed. A doctor was called, who gave her morphine. Three days later the same thing occurred in the left popliteal space, just as she was getting out of bed. This pain was also sudden and was a knife-like, terrorizing pain, so that she was unable to stand and fell back into bed. Three days later blueness began over the toes. The opinion was expressed that she had an embolus in either popliteal vessel; and in going back over the history we could interpret, or thought we could, why she had these emboli. She was a woman who was markedly sclerotic and had an auricular fibrillation of twelve years' duration. As with that condition thrombi not uncommonly form in the auricles, we believed that this had occurred with her. She had, then, embolized, first in one popliteal artery and three days later in the other. We were not quite clever enough to argue that the embolus had lodged temporarily at the bifurcation of the aorta, as a rider's thrombus, finally breaking, one portion going down and plugging the right popliteal artery, and later the other portion occluding the left. This was proved at necropsy.

I think from the 3 cases shown we must all recognize that the first thing in interpreting the physical signs is to have the guideposts with which to interpret them. The history gives us two of these: first, the leads or points that we should follow up in our physical examination; and second, it aids us to interpret correctly what we find.

Now if we take the other side we might say, "Well, if the history is so important let us take just the history and not dwell so much on physical findings." The case that has just been shown indicates the fallacy of that contention. The history and the physical examination are so closely interrelated that they cannot be separated at any time, although under some circumstances, either one separately may offer us most help.

Case IV. Female, aged forty-five.

History Summary. Simple childhood diseases; no complications; no severe illnesses in later life; never married. Last menstrual period September 27, 1922.

Present Illness began acutely one week ago with a chill of half an hour, then warmth. Twenty-four hours later sud-

denly seized with severe knife-like pain in right side, increased on deep breathing and coughing; eight hours later a dry, short, unproductive, painful cough; difficulty in breathing; later a mucus was expectorated and then a tenacious, glairy sputum. Herpes developed on the fourth day. Entered the hospital on the seventh day on account of increasing severity of all symptoms.

Now that is a fair history of lobar pneumonia. However, the physical signs showed she had a pericarditis. At necropsy the lung bed was completely negative and gave no evidence of a pneumonic or bronchopneumonic process. She had a purulent pericarditis. This case will serve as an example of many of a similar nature with which to emphasize the fact that one cannot rely on the history alone for a final interpretation of many cases.

These clinical cases have proved, I hope, that a careful history furnishes most important guides for the physical examination and the interpretation of its findings. The second great guide post in a thorough examination, is general inspection of the patient. And by general inspection I mean inspection of the entire body. The next case serves to demonstrate this point.

Case v. Female, married.

History Summary. Simple childhood diseases. Married at fifteen; three miscarriages, seven, two and three months in order named. Never pregnant again. Influenza at thirty, cough continued three months. Severe menorrhagia at thirty-three; fibroids discovered; operation July, 1920. Panhysterectomy.

Present Illness. Cough three months' duration, at first dry, then associated with slight mucus, never bloody; associated with dyspnea. Substernal pain and weakness. Shortness of breath two months' duration, worse on coughing, increasing. Lost 15 lbs. in two months. Appetite poor; feels generally weak.

That might not be a bad history of pulmonary tuberculosis, for instance. The individual was sent to me as one having pulmonary tuberculosis, but on examining her simply by inspection I found a prominence over the upper thorax which pulsated mildly, but visibly.

CASE VI.

Physical Examination Summary. Colored woman. Pupils equal, react slowly to light. Pulsation in suprasternal notch is marked. Prominence over upper thorax, 1st and 3rd interspaces; systolic shock over this. This prominence pul-

FIG. 114.

sates slightly. A_2 is greatly increased; short systolic bruit in aortic area; tracheal tug. Liver edge felt one finger's breadth below costal border—tender.

Clinical Opinion. Aortic aneurysm; luetic origin. Luetic hepatitis.

That inspection finding immediately stimulated us to investigate the case more carefully. When asked how long she had had it, she said, "Three months." When asked if it had decreased in size she answered, "No, I think it has increased a little." When asked if she had shown it to her doctor she said that he had not examined her. In this instance my point is that a general inspection examination would have brought out that one finding, which would pretty nearly make the diagnosis, because it would arouse enough interest to make one investigate it. Further examination showed an area of dullness on either side of the upper sternum, diastolic

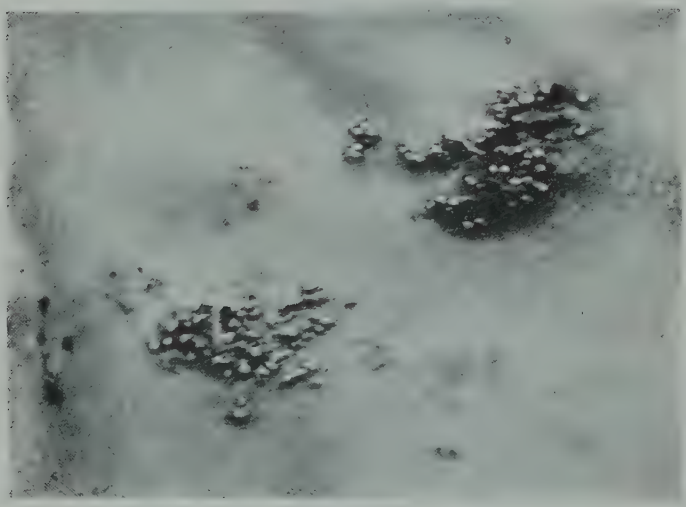


Fig. 115.

shock, and a tracheal tug, which led to a proper interpretation of aneurysm of the transverse arch.

Figure 115 is another example of the same type to emphasize the importance of a general inspection examination. This individual complained of pain beneath the right shoulder blade twenty-four hours prior to this picture. He had been to his doctor in the afternoon and told him that he had pain in that area. It was not associated with cough. It was not difficult for him to breathe. It was a burning, smarting pain, and the doctor gave him some capsules. That evening the

pain became more severe. On examination simple inspection revealed these areas of herpes zoster in the vessiculated stage, a crop of possibly twenty of them (these are only a few) along the sixth and seventh thoracic intercostal nerves on the right side. They would have been missed without the inspection examination.

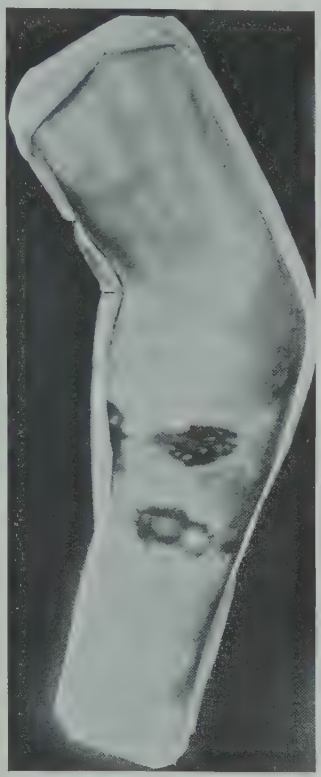


Fig. 116.

Case vii. To me Figure 116 is one of the most interesting I have to show because of the circumstances surrounding the patient.

This man was sixty years old. Four days previously he had been taken with chills, followed by temperature, and then he became comatose. The opinion was given that he had a central pneumonia, but as we examined him we were not satisfied with the lung bed findings as the explanation of his serious condition, and so we continued our examination downward over his body. The removal of his underclothing revealed nothing abnormal in his right leg, but as we went on and bared his left leg we found an area of erysipelas which had started from an old ulcer. This, then, was the real cause of his temperature, and his case is a definite demonstration of the value of a general inspection examination.

From the examples given, which have occurred in my experience in the last six weeks, the value of general inspection is obvious; it is one of the fundamental methods of examination, and we should use it conscientiously. But of course, in the thorough physical study of a patient it is not enough. The other methods—percussion, palpation and auscultation—should also be applied, for they commonly

unearth important findings that may not have been even surmised from the history or from the inspection examination.

The next case is one of many that could be cited to emphasize this point.

Case VIII. Female, married, aged sixty-one.

History Summary. Diphtheria twice while a child; no other illnesses. Married at forty-one; one miscarriage at three months unaccounted for; menopause at forty-two.

Fig. 117.

Failing eyesight for twenty years; in April, 1920, operated on at New York Eye Infirmary for glaucoma. Eyesight continued to fail, thinks poor sight accounts for inability to walk. Weakness in the legs and stiffness in the knees, gradually increasing for the last four years. Sensation of sitting on base balls when she is sitting. Becomes nervous and excited easily during the past two years.

This patient had four or five symptoms of such a severe nature that she did not mention three or four smaller ones that might have given us the clue. From the history one would be a little bit at sea as to the cause of her complaint.

But the physical examination added data that enabled us to interpret her case correctly.

Physical Examination Summary. Slightly thickened forehead skin; slight alveolar edge infection; right frontal and parietal region more prominent than the left; marked pulsation of the right carotid. A_2 is increased with a short systolic bruit; heart rate 96, regular; radial moderately sclerotic.

FIG. 118.

Blood pressure 200/90. Left femur is wider in the lower third than is the right; anterior outward curving and enlargement of the right leg, apparently bony; knee jerks absent on reinforcement; Achilles jerk absent; gait uncertain and tremulous.

Clinical Opinion. Paget's disease; arteriosclerosis; hypertension, apparently from renal sclerosis; paresthesia; absent knee jerks remain unanswered, possibly of luetic origin.

Figures 117 and 118 show the findings of the head and legs. Hence, in this case, the physical examination disclosed the condition of the head, the enlargement and curving of the tibiae and the absent knee jerks, as well as hypertension. It added, then, considerable data which had not been revealed by her history. From this data the opinion was expressed that she had Paget's disease. The absent knee jerks remained to be explained. The blood and spinal fluid were taken and both returned + + + + So in this case, the physical examination, when properly studied, allowed of the correct diagnosis of tertiary syphilis associated with an osteitis with Paget's syndrome.

II

The second division of this discussion deals with methods applicable to certain special examinations frequently not made, and with the interpretation of what they may reveal. The trend of medicine today is to depend so much on specialists that the practitioner has unfortunately discontinued the examination of certain very important special fields. I refer to the examination of the eyes, nose, throat, ears, larynx and pelvis.

Ordinarily when symptoms are present which point to these fields the patient is at once referred to a specialist. Let us reflect for a moment what the specialist does. If we send a patient to a gynecologist he examines the pelvis by looking and feeling; in other words he inspects and palpates the pelvis. The skin specialist inspects and arrives at his opinion. The otologist looks in the ear canal. It is easily seen that inspection is the most important factor of the specialist's work. As the methods for these investigations are not difficult and the equipment necessary is not costly, I feel that the general practitioner should become acquainted with and should include these examinations in his daily work. When definite disease is located in these special fields he can then refer his patient to the proper specialist for care.

I will start with a sinus case. The patient complained of headache, particularly over the right frontal region. It came on following influenza six months ago and had recurred many times especially when the patient had a "cold."

Apparently there was first an infectious element in the headache, and second a constant localization over the right frontal region. What should we learn from the physical examination of this area? If one thinks of a frontal sinusitis

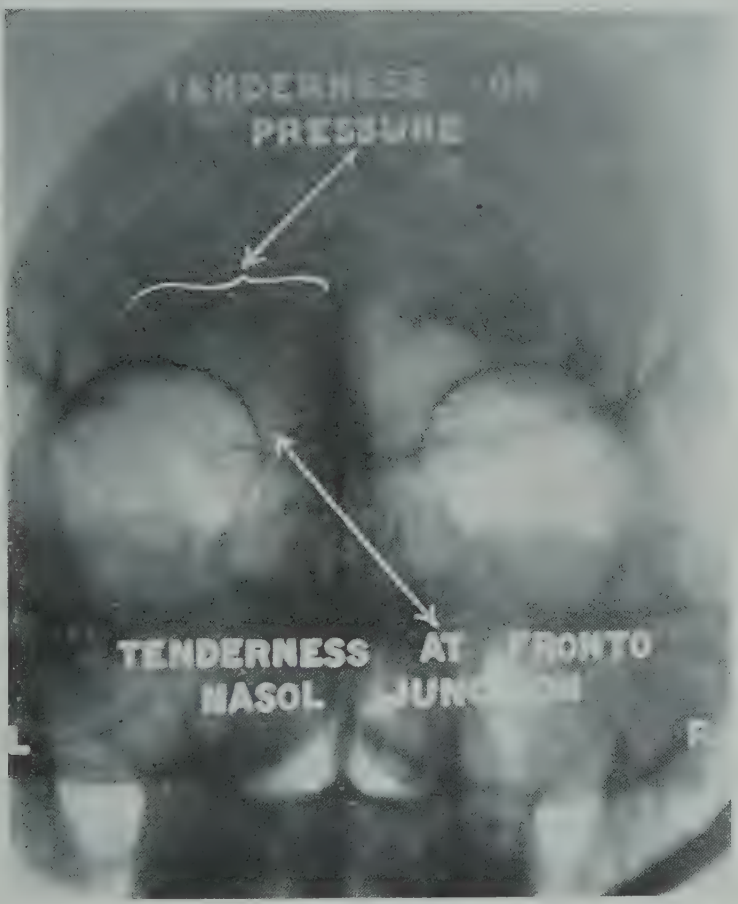


Fig. 119.

there are three things in the examination that any of us can do with a fair return of help for the effort. Frontal sinus disease has three fairly constant findings: pain, pressure and position. These can be remembered as the 3 P syndrome of frontal sinus disease. Pain or sensitiveness is produced by

pounding along the frontal region proper. Pain is also found as one presses on the nasofrontal junction or, if you like, as one presses up on the floor of the sinus from the upper orbit. Hence pressure is needed commonly to produce pain. The third factor, position, can be easily tested. If one lowers the head, making it a depending part of the body, and allows it to remain that way for a short time, the membrane of the sinus becomes congested and the pain or ache in that region is made definitely worse therefrom. Particularly is this true if the patient coughs or strains while in that position. By these simple methods this patient was interpreted as having a right frontal sinusitis, and this was confirmed by *x*-ray (Fig. 119).

Hence, by applying the simple methods described above, the cause of his headache was quickly discovered and the patient was at once referred to a specialist for proper treatment.

Quite a simple adjunct in examining frontal sinuses is the pocket flashlight. With the light placed below the orbital rim directed slightly upward and inward one can commonly outline the frontal sinuses in a very clear way. Even small variations of cloudiness in the two sinus regions can be discerned. The frontal sinus region, then, which is so commonly overlooked, and whose examination is frequently considered so difficult, can be reasonably well examined and interpreted by a physician in the field with the use of these simple procedures.

We will next take up the careful examination of the pupil of the eyes. It is easy enough, when there is a very large inequality of the pupils and they are light-stiff, to say that apparently the patient has a luetic infection, but we have certain conditions of the pupils which are more important, in percentage occurrence at least, than are light-stiff pupils; these are pupillary irregularity and slight inequalities.

Significance of Pupillary Irregularity and Inequality in Syphilis

Cases of above in series	86
Cases with syphilis	42
Cases with blood-Wassermann but with normal pupillary findings	164
Hence, percentage of cases of syphilis with pupillary signs	25

But 50 per cent of all cases of above were with syphilis

In connection with physical findings which might have a bearing on luetic infection we made a routine practice of taking the Wassermann reactions of both blood and spinal fluid in 1500 patients as they entered the Long Island College Hospital.

Out of the 1500 we found 86 had irregular or unequal pupils. Out of the 86, 42 had positive Wassermann reactions in either blood or spinal fluid, which made 50 per cent of all those with slight irregularity of the pupils having syphilis. By irregularity I mean a little incurving or a little infolding of the pupillary border, although the pupil still reacted. In other words, instead of being perfectly round in contour, it was irregular in outline.

Thus one quickly sees the reason for carefully examining the pupils for slight inequality and irregularity, an examination which is commonly elided, or the importance of whose slight findings is not properly interpreted. When we report that one out of every two patients with the findings mentioned had proved syphilis, its importance becomes self-evident.

We will continue with another case, which appeared with a complaint belonging to the realm of the specialist.

This individual had contracted a cold late in July (five months previous) and after that she had severe headaches, and for many nights was unable to sleep on this account. She had seen three doctors, one of whom had placed her on a diet, and the other two on medications. Her nose and sinuses had not been examined, although she had told her story of nose cold in July. In this case, simple nasal examination revealed marked enlargement of the turbinates pressing on the septum. It was felt from this finding that the headaches might well be caused by the pressure associated with nasal obstruction. Simple sinus examination by the methods above mentioned showed no evidence of sinus disease. She was sent to a rhinologist. As he used adrenalin on her nasal mucous membrane her headache stopped immediately, only to recur as soon as the subsequent dilatation appeared. Apparently this nasal condition was directly the cause of her complaint, as it had given rise to pressure on her sphenopalatine ganglion. When it was relieved she had complete recovery

from her symptoms.This calls attention to the fact that nasal examinations are not commonly made; as not one of her three physicians had examined her nose. This is another case in point where the examination is so simple and at times yields so much that it should be part of a general examination.

The next subject I will take up is inspection of the ear, another examination all too frequently passed over. Apparently its simplicity keeps the general practitioner from making it a part of his routine examination, even when necessary. Possibly this is due to his inability to interpret his findings. (Color slides of normal tympanic membrane and of acute suppurative otitis media not reproduced.)

These examinations of the sinuses, ears, nose and throat are so important at times that I feel we should all have the simple equipment that makes it easy to carry out the procedures. There is an electric outfit put up by Wappler and Company which can be used either in the office or in the field for this purpose.

Let us take another instance in which inspection is of the greatest importance. In the past it was difficult to say just what were the characteristics of diseased tonsils. After much bacteriological and pathological study correlating these with certain physical findings, we have arrived at the conviction that we can fairly definitely decide upon a pathological tonsil by its physical characteristics. It should be remembered that the tonsil does not have to be a large one to be diseased. The characteristics most valid with which to interpret pathological tonsils are: First, redness over the tonsil mucous membrane with the peritonsillar mucous membrane deeper in color than that of the surrounding mucous membrane. This simply means that there is a mild lymphangitis of the mucous membrane of the diseased area. The second point of importance is the presence or absence of glands of the neck. One would not ordinarily expect to find the neck glands in a normal person any more prominent than the axillary or inguinal glands. Hence if they are enlarged they are probably receiving drainage from some infection higher up. Enlarged glands become even more important as a sign if one has the opportunity of watching them increase and decrease at the time of an acute throat infection. The third point that aids in

detecting a diseased tonsil condition is to note whether the crypts are covered or contain exudate. The tonsils, then, that show redness more marked than the other mucous membranes of the throat, associated with neck glands, and with crypts that are covered or have exudate, are pathological tonsils, even though they may not be associated with acute attacks. I stress these points, knowing how important focal infection in this tissue is thought to be today in the interpretation and treatment of many general disease conditions.

Let us now pass from the examination of these special areas to the examination of the thorax, in which we will emphasize some of the short cuts that help in the study of the signs. There is a general rule in thoracic examination that admits of fairly exact interpretation, but is not often practised. If the examiner places his hands at corresponding points over the upper thoraces and finds they move equally; and again if both the axillary regions move alike on deep breathing, there can be no serious pleural or parenchymatous lung disease. That is, there cannot be present acute pleurisy, pleural effusion, thickened pleura, new growth in the pleura; or in the lung bed, pneumonia, moderately advanced tuberculosis, tumor of the lung, etc. These lesions are all associated with a lagging of the side involved during deep inspiration. Hence a finding which admits of such definite interpretation is worthy of general use. Again, in thoracic examination one often finds an upper area on one side not as full as that of the opposite side. It lags a little on breathing. The individual may show a clavicle a little more prominent on this side than on the opposite side. Such an asymmetry of form and respiratory excursion may be of significance, or it may not, depending upon the accompanying group of other physical signs. If with such asymmetry there is an increase in fremitus, and expiration is prolonged and high-pitched, that patient has fibrosis of that area. These physical signs tell us that the individual has had a destruction of the parenchymatous tissue of the lung in that area which has been followed by fibrous tissue formation. A fibrosis of this nature means an old lesion. But if râles are present with such physical sign its means that there is still an active pathology and that there

is an exudate there. These findings are so frequently present in the upper thorax that their reasonable cause might be dwelt upon for a moment with benefit.

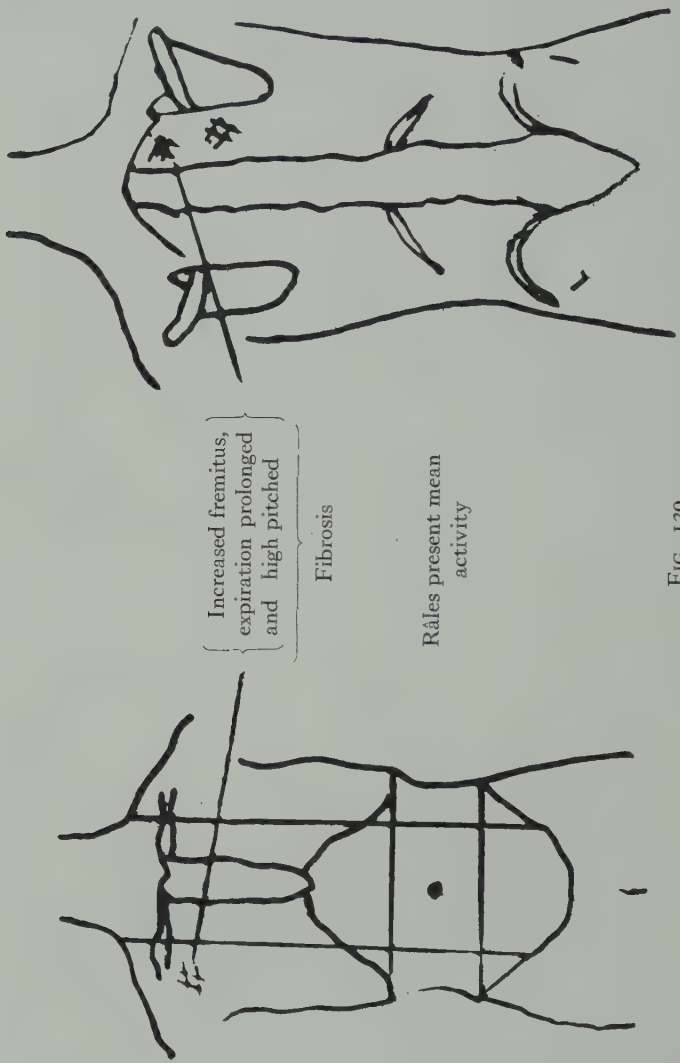


FIG. 120.

If we reflect for a moment on the common diseases of the upper lung we find that tuberculosis is found there out of proportion to anything else. As a matter of fact it is found so

commonly in the upper half of the thorax that it is known as a disease of the apices. Figures 120 and 121, taken from Garvin's data, are proof enough of the last statement.

If we take Garvin's data as a guide in interpreting physical findings of the upper thorax we find that 90 per cent of patients with incipient tuberculosis have their lesions in the lung apices. This justifies the statement that any group of physical signs, particularly of a chronic nature, over the upper portion of the lungs, either anteriorly or posteriorly, is reasonably due to tuberculosis, and should be so interpreted until we can prove that it is due to something else.

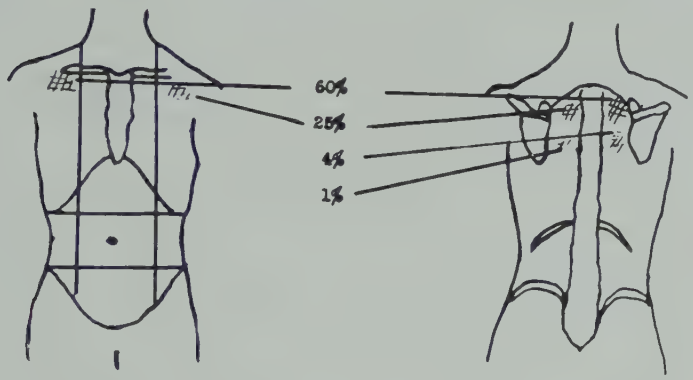


FIG. 121. Location of lesions of 500 patients with incipient tuberculosis.

On the other hand, it is equally true that physical signs over the lower halves of the lung bed are probably not due to a tuberculous lesion.

Another point I should like to emphasize, especially in thoracic examination, is a method of râle production. How frequently an individual is examined with a condition in which the examiner feels he should be able to elicit râles, and still on deep breathing only, there are none brought out. This statement is true, not only in examining the tuberculous but also in cases of beginning pneumonia and bronchopneumonia. But there is a method of bringing out râles, and hence bringing out signs of activity, in places where previously no activity was thought to exist. To do this the patient is instructed to take a deep breath and then expire

forcibly and deeply. At forced expiration he gives a single short cough. Commonly râles of a subcrepitating or crepitating character show following this short cough, or appear at the end of the next inspiration (Fig. 122).

As râles are the most important finding in the lung examination, it is equally important that the method to produce them is insisted upon. The patient takes a deep breath and then expires deeply and gives a single short cough. One should not accept a grunt or strain. The cough should not be too loud, for in that case small râles which may follow it will be made inaudible. These few points in examining the lung have such wide application and admit of such definite

FIG. 122. Râles are best brought by *cough*—and appear just at end of *expiratory* cough or at end of following inspiration.

interpretation that they merit a place of great importance in routine examination.

Let us now pass onward to a few fundamental points in the examination of the heart and to their interpretation. We all know that the apex impulse is an important guide to the size of the heart, but we very often fail to look for it. If we could do but one thing in the examination of the heart, apparently the most important thing would be to locate the apex impulse. To prove the value of this as an aid in determining the heart size and position I selected patients, irrespective of their diseases, and studied by the use of the teleoroentgenogram the relation of apex impulse to the left heart border. The apex impulse was carefully localized in each case and a lead cross placed directly over the punctum maximum. The teleoroentgenograms were then taken. Figure 123 demonstrates the point. Of all the patients so studied the apex impulse as determined by inspection never varied more

than 1 cm. from the left border as determined by the teleoroentgenogram.

In addition to this short cut in determining the left border of the heart there is a simple method applicable to determine

Fig. 123.

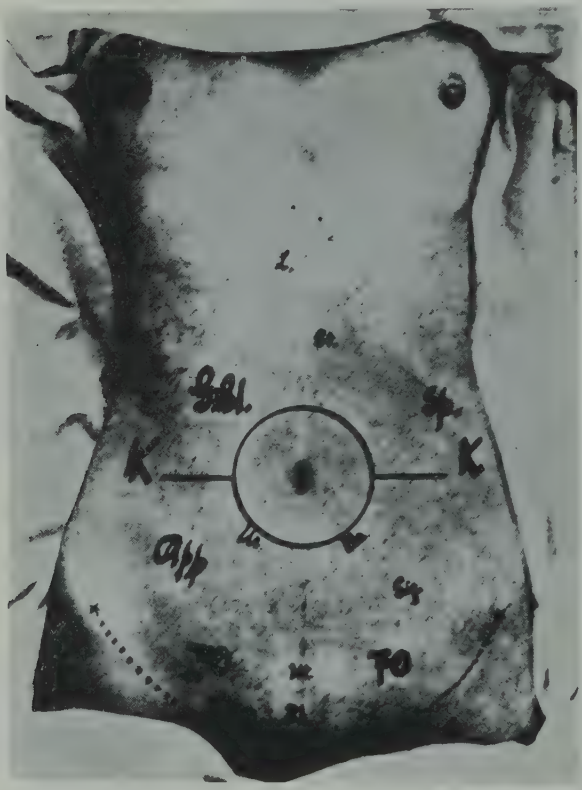
the capacity of that organ. Each year this test has been tried on my students to determine their reactions. The results obtained on them have been considered as showing normal variations.

Exercise Test for Heart-rate Response in 100 Students

From sitting to standing position—Increase in heart rate, 0–9.

Increase after squatting 10 times, 8–36. All returned to normal between two and three minutes.

Increase after hopping 100 times, 14–50. Returned to standing rate in four minutes.



Four points noted in abdominal examination.

1. Localized rigidity.
2. Localized tenderness.
3. Organ edges;
   (*a*) Liver.
   (*b*) Spleen.
   (*c*) Kidneys.
4. Masses.

Fig. 124.

In 100 students the heart rate increased from 0 to 9 beats a minute when they stood erect from a sitting position. Many individuals showed no increase in rate. Only 2 in the 100 increased their rate 9 beats. But these men did not have heart disease. Hence, as an index, we have a variation from 0 to 9 beats a minute as normal reaction to this change of position. On the other hand, individuals who have myocar-

dial insufficiency commonly show an increase of from 12 to 36 beats a minute by this simple postural change.

The exercise test is carried farther in having the students assume a squatting position ten times. This brings about a much greater change in rate. Among my students the rate increased from 8 to 36 beats a minute. All returned to normal in two and a half minutes.

It must be borne in mind that at times it is difficult to get a positive idea as to heart capacity by such simple procedures; and this is beause the heart muscle in different individuals has a tonicity of different degrees, depending upon how much that organ has been asked to exercise. If it has not been exercised much, the tone necessarily is poor and the response to the exercise test will be rather extreme, even when no cardiac disease is present. Again, it is to be remembered that there is a reserve power in the heart that is used only at times of extra effort, and if an individual has no reserve power and little tone, as is seen in certain myocardial diseases, then the heart rate, increased by exercise, continues over a long period of time, because the reserve is not there to reclaim its slower rate. Thus it will be seen that each case will be interpreted through its own response, bearing the above in mind; but it can also be seen that simple exercise tests can yield information concerning the heart's muscular efficiency.

Passing from these simple methods which can so easily be applied to the examination of the heart, and which I feel are of such value, I shall comment for a moment upon the routine examination of the abdomen.

In abdominal examination we should keep certain points in mind if we are to cover this part thoroughly. First, localized rigidity should be looked for by very light and then by moderate palpation. With this any localized tenderness should be noted. The entire abdomen should be palpated, always comparing the corresponding right and left areas. The localization of either rigidity or tenderness must be interpreted with other findings. Figure 124 shows the organs most commonly involved when rigidity or tenderness are found in the areas indicated. The next point in this examination is the search for organ edges; that is, specifically feeling for the liver edge, splenic edge and kidney poles.

The abdomen should then be palpated for the possibility of masses. The description of such findings and their interpretation depending on localization cannot be dwelt upon here. If one goes over the abdomen in such a simple and thorough-going manner he often runs across findings that would not come to light by a more superficial observation.

It might well be said that in examining an individual by these fundamental methods he has been covered from head to foot, and that a number of physical findings may have been unearthed which apparently do not bear upon the clinical complaint of the individual. That contention is true. Frequently more disease is found than we anticipated from the history, and some of the conditions discovered may not have any bearing upon the patient's complaint. However, correcting diseased states before they influence the general economy of the patient is certainly a definite function of the physician. In other words, a thorough routine examination gives the patient an inventory of his physical status. Hence a detailed general examination justifies itself, because only by this method can a physician offer his best opinion and so direct the patient along proper paths; and thus also may he guide the patient toward preventive medicine.

After the physical examination is completed a brief summary of the positive findings should be made, so that the chaff may be separated from the wheat.This summary helps to clarify the mind of the examiner. Not infrequently as one puts down the positive findings in this summary he finds that he is able to express a reasonable clinical opinion of the case, although he had felt unable to do so from the unrelated facts noted as he went along.

In this brief paper I have attempted to accent the importance of the clinical history as the most noteworthy guide to a proper physical examination and to the interpretation of the findings of that examination. A clinical history should yield us more than any one other factor in the examination when it comes to solving the problem. Second, I have tried to present the simplicity with which many commonly omitted examinations may be carried out. I refer to the examination of the sinuses, ears, nose and eyes. Further, some simple methods for every-day use in the examination of the lungs,

heart and abdomen were touched upon. Although I realize how incompletely I have dwelt on all these subjects, I have purposely planned to stress only a few of the points, so easy to overlook, which can be used daily by the physician in the field, and which are of such help to all of us when made a part of our regular routine.

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